‘A’ Level Sport Studies

Teaching Support Pack

Images and Performance Data for Exercise Physiology

Colin Clegg

Lecture Notes

FELTHAM PRESS

ISBN 1-902265-05-X

Images and Performance Data for Exercise Physiology

by Colin Clegg

Contents

Introduction

Units

Accessing and Analysing Elite Performance Data

Image 1 100 metres Olympic Finals

Image 2 200 metres

Image 3 400 metres World Records *(Men)*

Image 4 800 metres World Records *(Men)*

Image 5 Mile World Records

Image 6 5 000 metres World Records *(Men)*

Image 7 10 000 metres *(Men)*

Image 8 Marathon *(42 k or 26.2 miles)* Men

Image 9 Differences Between Men's and Women's Performances

Continued....

Contents

Image 10 Energy from ATP

Image 11 ATP Regeneration from Creatine Phosphate

Image 12 Skeletal Muscle Myofibrils

Image 13 Slow and Fast Twitch Muscle Fibres in Humans

Image 14 Direct Observation of Slow & Fast Twitch Muscle Fibres in other animals

Image 15 Effect of Endurance Training on Slow Twitch Muscle Fibres

Image 16 Muscle damage and delayed onset muscle soreness

Image 17 Effects of Endurance Training and Doping on Blood Composition

Image 18 Capacities of the ‘Normal’ and ‘Athlete’s’ Heart of Subjects Matched for Size and Age

Image 19 Nature or Nurture - Twin Studies

Image 20 Horses for Courses

Appendix 1 Photocopiable Sheets 1-20

Appendix 2 Session Overhead Transparencies 1-20

Introduction

These lecture notes will help enliven the presentation of exercise physiology.

Students without a background in biology often tend to find the subject of exercise physiology difficult, particularly if the subject is presented as a modified version of human biology. The plethora of descriptive terms and definitions, basic chemical and physical concepts and much more, tend to alienate new comers to the subject before any connection can be made to their intrinsic interest in PE and Sport Studies.

Experience has shown that by taking a performance based approach, interest and enthusiasm can be kindled, before any realisation that a subject known as exercise physiology is being studied.

Once the realisation has dawned, then there is a need to provide the hard pressed teacher with some stimulating focus points to maintain the interest.

All parts of the subject do not lend themselves to this approach, and the material presented here is selective, and also limited by the size *(and cost)* of the pack. However, it is hoped that once familiar with the approach, teachers will be able to build on this base, year by year.

Some of the material may be considered to be over and above that which is strictly required by most students at this level, but such material has the important role of broadening the hidden base of that ‘iceberg’ of understanding, the tip of which appears as good confident teaching in the classroom, and which is always threatening to melt in the ‘gulf stream’ of developing research and changing syllabuses.

Whilst recognising the need for clarity of organisation and presentation, the seamless nature of the subject renders it difficult to avoid interconnections, and once a certain level of confidence is gained such interconnections can be very useful in bringing things together. Thus there is much common ground throughout these notes, and students should be encouraged to explore these links themselves.

The data is presented in a variety of ways to illustrate some of the different ways in which they might be analysed, and students should be encouraged to devise their own ways of presenting them.

Naturally the data provided for the various running events will become dated, but the value of the physiological insights they offer will remain unaltered. If after a period it is felt that they should be updated, then this in itself presents useful opportunities for enquiry.

The Notes provide the key information on each image. The black and white miniatures of the images in the Notes, link the Notes to the A4 black and white and A4 full colour versions of the Images. However, if enlarged sufficiently the miniatures will reveal full details. The A4 black and white versions are for ease of printing, including onto acetate for OHTs. The A4 full colour versions are for on-screen use, projection, and printing onto acetate for OHTs.

Units

Special Note on units of volume

The litre *(l)* and millilitre *(ml)* are still widely used in exercise physiology, but they are not SI units, and the symbol for litres *(l)* can cause confusion eg 1.0 l. The System Internationale *(SI)* of units is intended to be used universally. Therefore the terms 'decimetre cubed' *(dm^3)* i.e. 10 cm x 10 cm x 10 cm *(1000 cm^3)* and 'centimetre cubed' (cm^3) are the more consistent and are used in all sciences, as they are here. The persistence of the litre in exercise physiology could be explained by the extensive involvement with non-science trained athletes and coaches, to whom the term decimetre cubed would be unfamiliar compared to the litre which is in common use in everyday life. A situation rather akin to the persistence of the calorie as a unit of energy in the face of the more scientifically correct SI unit the joule.

Special Note on units of oxygen uptake

Oxygen uptake is shown as VO_2. The units used can be cm^3 per minute, and may be quoted per kilogram of body weight. This can be shown as VO^2 $cm^3.min^{-1}.kg^{-1}$. Another convention is to use a dot over the V to indicate 'per minute' eg. $\dot{V}O_2$ $dm^3.kg^{-1}$ thus removing the min^{-1} from the units

Special Note on units of heart rate

Heart rate is measured as beats per minute. This is frequently and more familiarly shown as 'bpm', and more correctly as $b.min^{-1}$. Both are used here.

Accessing & Analysing Elite Performance Data

Accessing Performance Data

Elite performance data is readily available from specialised magazines such as Athletics Weekly, Cycling Weekly, Cycle Sport etc. In Athletics the National Union of Track Statisticians *(NUTS)*, an entirely voluntary organisation, is the ultimate source of such information in GB. Similarly the IAAF *(and the equivalent bodies for many other internationally recognised sports)* publish Year Books which are authorative sources, and usually readily available. For those with access, the Internet is an increasingly important source.

Analysing Performance Data

Some elite athletic performance data has been selected and analysed in a number of ways. Athletic events lend themselves particularly well to this approach as a result of the 'simplicity' of their demands on the body systems.

An analysis of running performances throughout the whole range of competitive distances illustrates many interesting physiological principles.

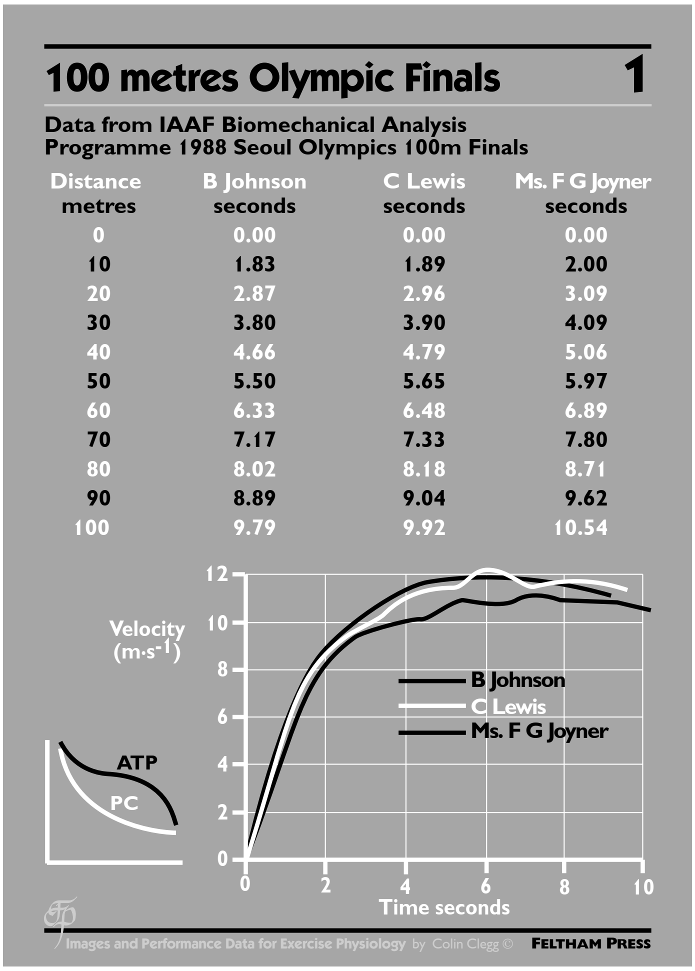
The data can be plotted in any number of ways, and each different choice of axes should reveal fresh insights into the performance and its underlying physiology. In these notes the data has been plotted in several different ways, not because they are the 'best' way of presenting the data, but simply to illustrate a range of different approaches, which is by no means exhaustive.

As with all events, the pattern of performance is virtually dictated by the underlying physiology - which is the main theme of this approach. Once it is clearly seen that sport performance is the expression of the underlying physiology, then students should be well motivated to study that physiology.

Furthermore, an understanding of the superlative elite performances should induce a deeper appreciation of the functioning of the body systems to be studied.

100 metres Olympic Finals

Image 1



100 metres Olympic Finals 1

Data from IAAF Biomechanical Analysis Programme 1988 Seoul Olympics 100m Finals

Distance metres	B Johnson seconds	C Lewis seconds	Ms. F G Joyner seconds
0	0.00	0.00	0.00
10	1.83	1.89	2.00
20	2.87	2.96	3.09
30	3.80	3.90	4.09
40	4.66	4.79	5.06
50	5.50	5.65	5.97
60	6.33	6.48	6.89
70	7.17	7.33	7.80
80	8.02	8.18	8.71
90	8.89	9.04	9.62
100	9.79	9.92	10.54

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

The levelling off, and/or dropping off, of the velocity/time curve can be interpreted on the basis of ATP/CP depletion in flat out sprint activities.

Although all energy systems contribute at any one time, the ATP/PC or phosphagen system supplies most of the energy in the first few seconds, with an increasing contribution from the anaerobic lactic system towards the latter part of the event.

The transition from one majority system to another raises discussion about the concept of 'physiological thresholds'.

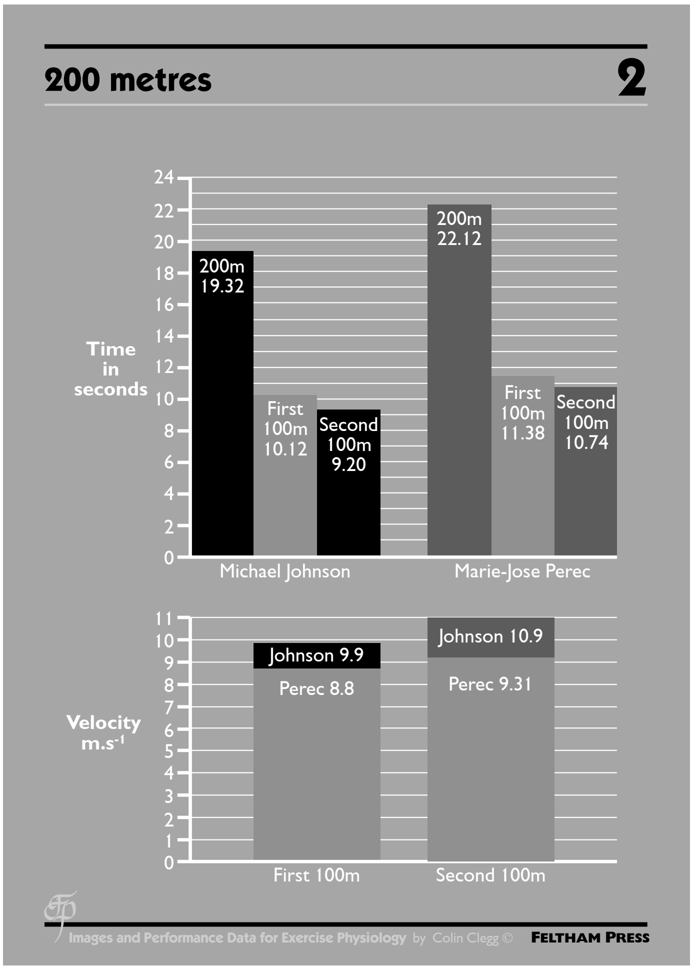
The untimely death of Ms F G Joyner in 1998 at the age of 38, raised fresh speculations about drug abuse, but the autopsy revealed no evidence of such.

Interesting background points for discussion behind these performances are that; Ben Johnson was disqualified and banned as a result of a positive test for steroids, and that quite amazingly from a 'nature versus nurture' point of view, two out of the first three were Jamaican born *(Johnson(1st) running for Canada, and Christie (3rd) running for Britain)*.

Donovan Bailey's 10 m splits in his World Record 9.84 s winning run in the 1996 Atlanta Olympics were: 1.9, 1.2, 1.0, 0.8, 0.7, 0.9, 0.9, 0.8, 0.8, 0.84; reaching a maximum speed of 12.1 metres per second *(27.1 mph)* just before the 60 metres point.

200 metres

Image 2



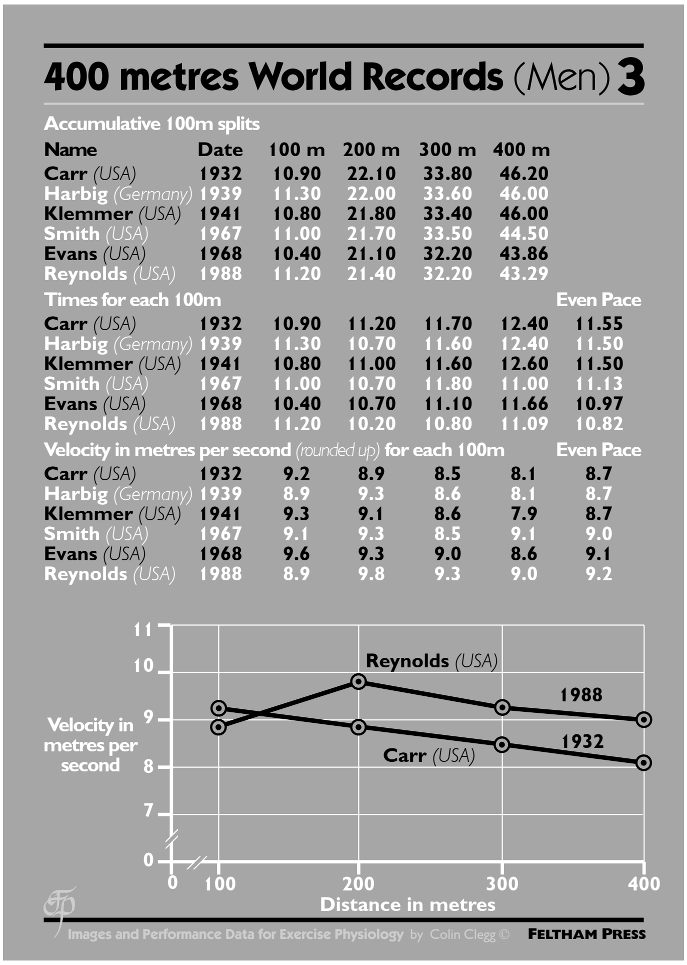
Compared to the 100 m the 200 m increasingly involves the lactic anaerobic energy system. The event is complicated by the bend running in the first half of the race, with each lane having a different angle of curvature, and much being made of the 'psychological' advantage of running on a lane inside one's main rival*(s)*. This also results in problems of gathering time data at 10 m intervals. The event 'splits' usually only being provided for the first and second 100 metres.

Michael Johnson's 100 m splits when he set a World Record of 19.32 s at the 1996 Atlanta Olympics were 10.12 s and 9.20 s The second half being 0.92 s faster than the first as a result of the 'flying start', despite the accumulating lactic acid and depletion of phospho-creatine.

At the same Olympics, in the women's 200 m Marie-Jose Perec ran 22.12, with splits of 11.38 and 10.74, a difference of 0.64 s.

400 metres World Records *(Men)*

Image 3



400 metres World Records (Men) 3

Accumulative 100m splits

Name	Date	100 m	200 m	300 m	400 m
Carr *(USA)*	1932	10.90	22.10	33.80	46.20
Harbig *(Germany)*	1939	11.30	22.00	33.60	46.00
Klemmer *(USA)*	1941	10.80	21.80	33.40	46.00
Smith *(USA)*	1967	11.00	21.70	33.50	44.50
Evans *(USA)*	1968	10.40	21.10	32.20	43.86
Reynolds *(USA)*	1988	11.20	21.40	32.20	43.29

Times for each 100m

Name	Date	100 m	200 m	300 m	400 m	Even Pace
Carr *(USA)*	1932	10.90	11.20	11.70	12.40	11.55
Harbig *(Germany)*	1939	11.30	10.70	11.60	12.40	11.50
Klemmer *(USA)*	1941	10.80	11.00	11.60	12.60	11.50
Smith *(USA)*	1967	11.00	10.70	11.80	11.00	11.13
Evans *(USA)*	1968	10.40	10.70	11.10	11.66	10.97
Reynolds *(USA)*	1988	11.20	10.20	10.80	11.09	10.82

Velocity in metres per second *(rounded up)* for each 100m

Name	Date	100 m	200 m	300 m	400 m	Even Pace
Carr *(USA)*	1932	9.2	8.9	8.5	8.1	8.7
Harbig *(Germany)*	1939	8.9	9.3	8.6	8.1	8.7
Klemmer *(USA)*	1941	9.3	9.1	8.6	7.9	8.7
Smith *(USA)*	1967	9.1	9.3	8.5	9.1	9.0
Evans *(USA)*	1968	9.6	9.3	9.0	8.6	9.1
Reynolds *(USA)*	1988	8.9	9.8	9.3	9.0	9.2

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

The 400 m produces the greatest accumulated oxygen deficit *(AOD)* of all track events. As with the 200 m there are problems associated with bend running, and the time data is usually only available at the 200 m marks.

A too fast start will always 'find the athlete out', as the levels of lactic accumulation are such that the pace over the last 50 m can be catastrophically slow, as athletes 'tie up' before the finish line. As with the 800 m, this can be a great event for 'strong' finishes in which the athlete which is slowing the least appears to come with a surge at the end.

Unlike the 200 m, the second half of the 400 m has never been run faster than the first half in a Championship event, a dramatic illustration of the overwhelming effect of the accumulation of lactic acid.

The above events have been selected from a total of 24 World Record runs since 1928, as they are the only ones for which 100 m splits have been published.

Electronic timing, allowing the timing of events to 1/100 second, was introduced in 1968.

Continued....

Apart from Harbig in 1939, Reynolds' first 100 m is the slowest, but his second, third, and fourth are easily the fastest compared to all previous World Record runs. *(Reynolds was subsequently involved for years in a bitter legal dispute with the sport's authorities over dope testing results.)*

Harbig also ran a ground breaking 1 min 46 secs in the 800 m, under the guidance of the new 'scientific' interval training based on heart rates after effort and recovery, of Gerschler and Reindell, which are still in use today. Only Jesse Owens was more highly regarded in the 1930's. Harbig ran 50 races at 400 m over his career, winning 45 of them. He remains the only man to have set World Records from 400 m to 1000 m. Harbig's 100m splits were 11.3,10.7, 11.6, 12.4, giving him a first 200 m split of 22.0, at which point he was led by Lanzi in 21.7, however Lanzi faded, running the last 100 m 1.3 s slower than Harbig who overtook him just after the 300 m mark. Harbig was killed in the Second World War on March 5th 1944.

Lee Evans time was set at an altitude of 2248 m *(7000 feet)* above sea level in Mexico City in the 1968 Olympic Final, and his first 100 metres is the fastest of all time. The electronic time for this race was accepted as the first IAAF automatically timed world record. This sensational time stood for 20 years until bettered, without the advantage of the thin air of altitude, by Reynolds in Zurich in 1988.

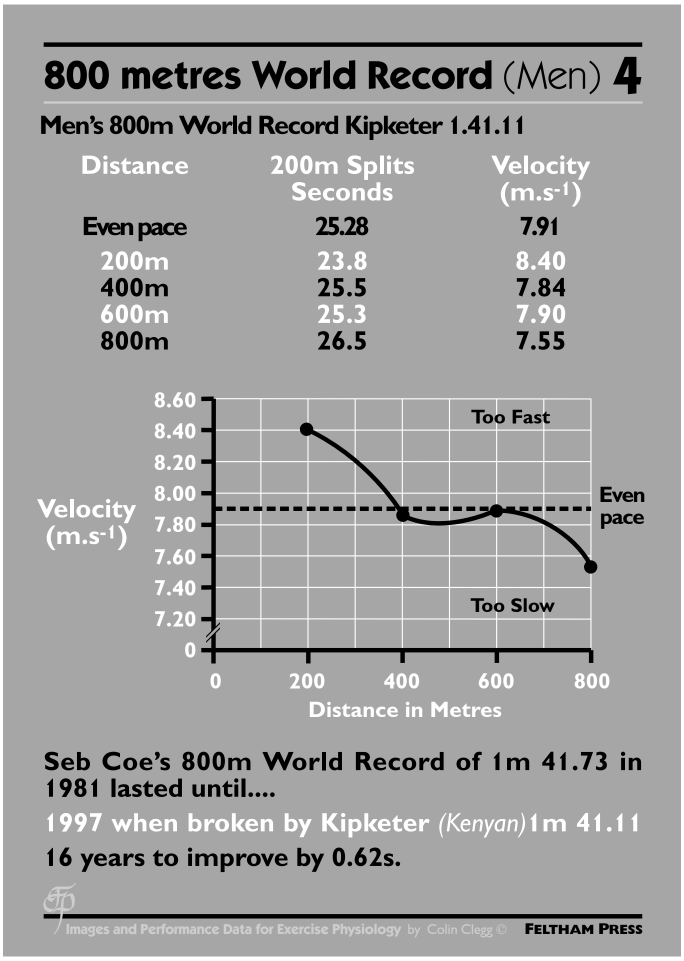
Lee Evans' run at altitude illustrates the anaerobic nature of the 400 m, where a relative lack of oxygen in the thin air of altitude has no deleterious effect.

The fastest first 200 metres of 20.9 was run by both McKenley and Rhoden of Jamaica in 1950, but interestingly they also then both ran the slowest second 200 metres of 24.9 since 1932.

In earlier times the 440 yards has been raced on non standard tracks, including those with a straight first 220 yards, and even a completely straight 440 yards track. This obviously had implications for the timings, not only with regard to the faster speed when running in a straight line, but also with regard to the overall slope and wind direction. For example, Herb McKenley of Jamaica ran a wind assisted 45.0 on a straight track in 1947 when his World Record on a standard track stood at 46.3. In the period when 'Imperial' distances *(yards and miles)* were regularly run *(by athletes competing in the English speaking countries, especially the USA, Great Britain and its then Empire/Commonwealth)*, the times in the 440 yards were converted to 400 m equivalents by subtracting 0.26 s.

800 metres World Record *(Men)*

Image 4



800 metres World Record (Men) 4

Men's 800m World Record Kipketer 1.41.11

Distance	200m Splits Seconds	Velocity (m.s^{-1})
Even pace	25.28	7.91
200m	23.8	8.40
400m	25.5	7.84
600m	25.3	7.90
800m	26.5	7.55

Seb Coe's 800m World Record of 1m 41.73 in 1981 lasted until....

1997 when broken by Kipketer *(Kenyan)* **1m 41.11**

16 years to improve by 0.62s.

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

The 800 m has increasingly become a speed/endurance event, albeit somewhat tactical.

Theoretically, the most efficient way physiologically of running any event above a pure flat out sprint, is to run it at even pace. Bursts of faster than even pace running, other than for tactical reasons, are considered inefficient as they would involve the lactic anaerobic system. This leads to the accumulation of lactic acid, resulting in pain and fatigue, and depletes the muscle glycogen stores *(although the latter would only affect performance in the longer events)*.

In the 800 m the relative involvement of aerobic to anaerobic energy systems is estimated at 57% aerobic to about 43% anaerobic, with about 5% of the anaerobic component coming from the ATP/PC phosphagen system.

In the event shown above the fast first 200 m would have been partly tactical and partly psychological, but inevitably results in lactic acid production.

Continued...

The accumulation of lactic acid forces a reduction in the pace, so that the aerobic system can increasingly contribute to the energy demands, with a corresponding decrease in lactic acid production and possibly some oxidation of lactic acid.

The third 200 m was at virtually even pace, and Kipketer will have been gathering himself for the final flat out effort of the last 200 m, where all systems would be taken to the limit, culminating in depletion of the ATP/PC system and the accumulation of lactic acid to critical levels, bringing the performance to a virtual standstill just beyond the finish line.

Thus the 800 m is an excellent event for involving performance based considerations of the interplay of energy systems.

It is also interesting to speculate what an even pace performance would have appeared like if an opponent had run an even pace of 25.27 s in the same race as Kipketer.

At the end of the first 200 m our 'ghost' runner would have trailed by 1.47 s or about 11.6 metres. At the bell he would have closed by only 0.23 s or about 1.8 metres, still being 9.8 metres down. By the start of the last 200 metres he would have closed by another 0.03 s or about 0.2 metres, still 9.6 metres down. However, over the last 200 metres he would have appeared to come with a tremendous burst of speed to win on the line in 1m.41.08s. The perfect 'steady state' run requiring superhuman control and judgement. Although it must be said that many have the experience of starting 'slow' and never being able to 'catch up'.

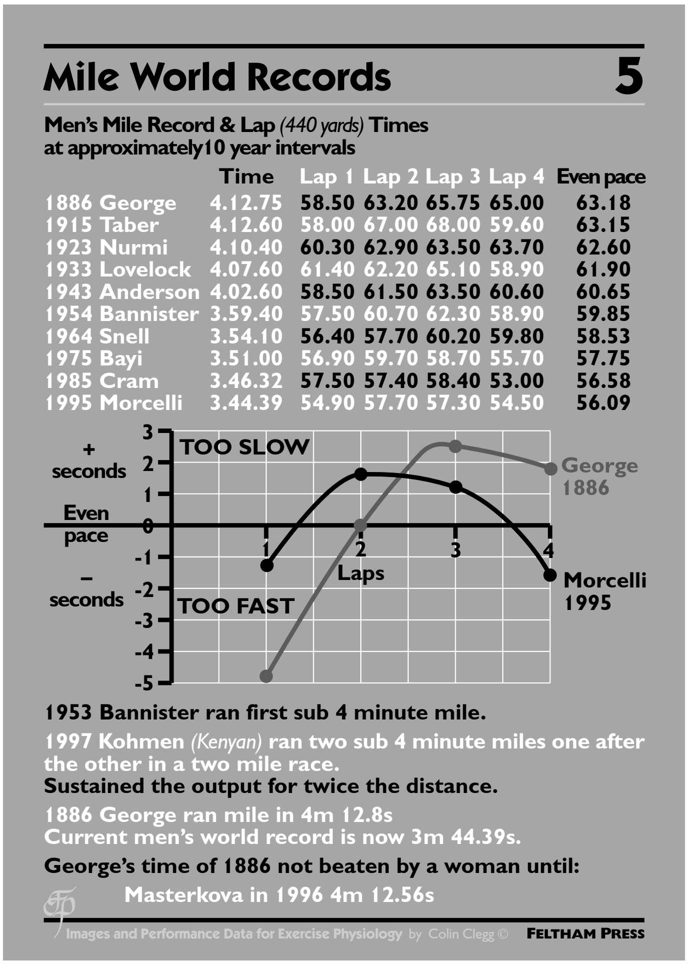
Harbig's 1:46.6 in 1939 *(on a loose cinder 500 m track),* is considered to be one of the most dramatic improvements to have been made in the history of athletics, some would consider it equivalent in impact to Bob Beaman's famous long jump in the 1968 Mexico Olympic Games. It was an excellent example of even-paced running with 200 m splits of 26.0, 26.8, 27.0, and 26.8. This record lastest for 16 years until 1955. In 86 races at 800 m Harbig lost only 12 *(including 4 heats where winning was of no importance)*. At one point he had won 50 consecutive races at the distance.

As mentioned in the discussion of the 400m, Harbig was the first exponent of Gerschler and Reindell's 'scientific' interval training, based on monitoring of heart rates during alternating effort and recovery 'intervals, which is still a fundamental tenet of coaching and training today.

The victory of Barthel in the 1952 Helsinki Olympic Games 1500 m, another Gerschler and Reindell athlete, established their international reputation, and many athletes sought their advice and guidance. British athlete Gordon Pirie *(a future World Record holder at 3000m and 5000m)* received criticism from some quarters in Britain for consulting German coaches. The eventual widespread acceptance and durability of their methods illustrates the universal nature of sporting achievement.

Mile World Records

Image 5



Mile World Records

5

Men's Mile Record & Lap *(440 yards)* **Times at approximately10 year intervals**

	Time	Lap 1	Lap 2	Lap 3	Lap 4	Even pace
1886 George	4.12.75	58.50	63.20	65.75	65.00	63.18
1915 Taber	4.12.60	58.00	67.00	68.00	59.60	63.15
1923 Nurmi	4.10.40	60.30	62.90	63.50	63.70	62.60
1933 Lovelock	4.07.60	61.40	62.20	65.10	58.90	61.90
1943 Anderson	4.02.60	58.50	61.50	63.50	60.60	60.65
1954 Bannister	3.59.40	57.50	60.70	62.30	58.90	59.85
1964 Snell	3.54.10	56.40	57.70	60.20	59.80	58.53
1975 Bayi	3.51.00	56.90	59.70	58.70	55.70	57.75
1985 Cram	3.46.32	57.50	57.40	58.40	53.00	56.58
1995 Morcelli	3.44.39	54.90	57.70	57.30	54.50	56.09

1953 Bannister ran first sub 4 minute mile.

1997 Kohmen *(Kenyan)* **ran two sub 4 minute miles one after the other in a two mile race.**
Sustained the output for twice the distance.

1886 George ran mile in 4m 12.8s
Current men's world record is now 3m 44.39s.

George's time of 1886 not beaten by a woman until:
Masterkova in 1996 4m 12.56s

Images and Performance Data for Exercise Physiology by Colin Clegg © **FELTHAM PRESS**

Although not a metric distance, the mile has remained a 'classic' distance, the world record of which is still keenly contested.

The progression of the record by 28.36 s since 1886 represents an 11.2% improvement, a distance at 1886 pace of about 198 yards, or at 1993 pace of about 222 yards, ie about half a lap.

The relative demands of the whole event on the energy systems is estimated at about 76% aerobic and 24% anaerobic.

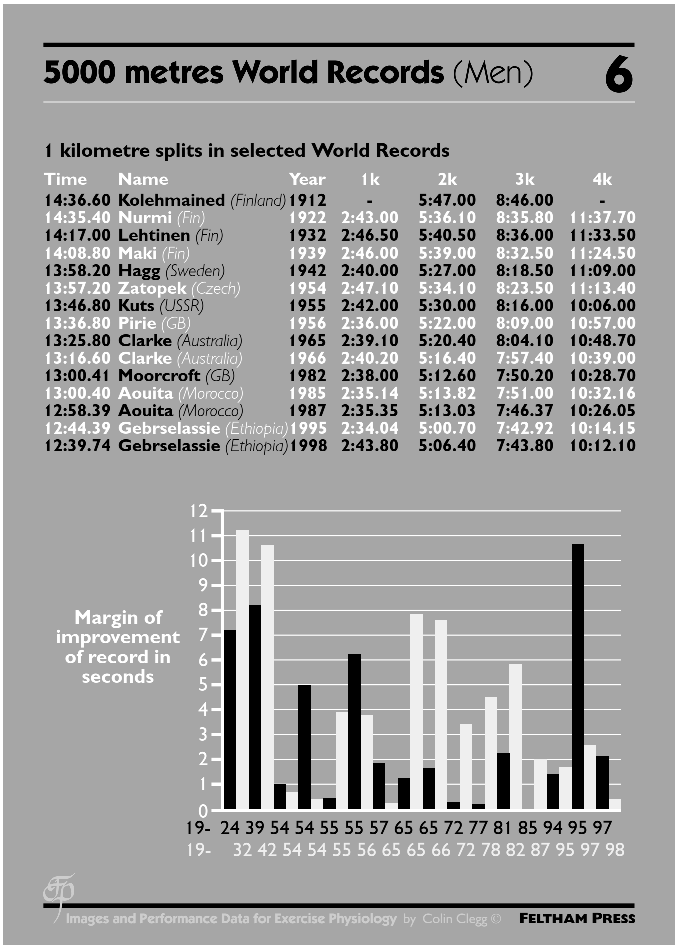
George's first lap is clearly much too fast and must have resulted in a large lactic acid accumulation, a conclusion reinforced by the drop off in pace in laps 2 and 3.

Morcelli's performance appears much more controlled, no doubt as a result of the years of accumulating wisdom of the efficiency of even pace running, and the fact that the faster the even pace required, the more difficult it is to run "too fast' relative to it. It is interesting to note that Morcelli never varied from either side of even pace by more than about 2.8%, whereas the figure for George is 7.4%.

The disparity between men's and women's performances over the mile acts as an interesting source of debate about gender differences in sport.

5000 metres World Records *(Men)*

Image 6



5000 metres World Records (Men) **6**

1 kilometre splits in selected World Records

Time	Name	Year	1k	2k	3k	4k
14:36.60	**Kolehmained** *(Finland)*	1912	-	5:47.00	8:46.00	-
14:35.40	**Nurmi** *(Fin)*	1922	2:43.00	5:36.10	8:35.80	11:37.70
14:17.00	**Lehtinen** *(Fin)*	1932	2:46.50	5:40.50	8:36.00	11:33.50
14:08.80	**Maki** *(Fin)*	1939	2:46.00	5:39.00	8:32.50	11:24.50
13:58.20	**Hagg** *(Sweden)*	1942	2:40.00	5:27.00	8:18.50	11:09.00
13:57.20	**Zatopek** *(Czech)*	1954	2:47.10	5:34.10	8:23.50	11:13.40
13:46.80	**Kuts** *(USSR)*	1955	2:42.00	5:30.00	8:16.00	10:06.00
13:36.80	**Pirie** *(GB)*	1956	2:36.00	5:22.00	8:09.00	10:57.00
13:25.80	**Clarke** *(Australia)*	1965	2:39.10	5:20.40	8:04.10	10:48.70
13:16.60	**Clarke** *(Australia)*	1966	2:40.20	5:16.40	7:57.40	10:39.00
13:00.41	**Moorcroft** *(GB)*	1982	2:38.00	5:12.60	7:50.20	10:28.70
13:00.40	**Aouita** *(Morocco)*	1985	2:35.14	5:13.82	7:51.00	10:32.16
12:58.39	**Aouita** *(Morocco)*	1987	2:35.35	5:13.03	7:46.37	10:26.05
12:44.39	**Gebrselassie** *(Ethiopia)*	1995	2:34.04	5:00.70	7:42.92	10:14.15
12:39.74	**Gebrselassie** *(Ethiopia)*	1998	2:43.80	5:06.40	7:43.80	10:12.10

Images and Performance Data for Exercise Physiology by Colin Clegg © **FELTHAM PRESS**

The early dominance of the ‘Flying Finns’ is remarkable, and was based on Pikhala’s ‘terraced’ training methods, a forerunner of the interval training of Gerschler and Reindell in Germany,

Hagg and Anderson of Sweden, advocates of ‘fartlek’ or ‘speed play’ over natural terrain, would almost certainly have made bigger inroads into the record books at several distances, were it not for the Second World War, including possibly the sub-4 minute mile - to which they were very close, more than a decade before it actually went in 1953.

Zatopek is one of the enduring names in Olympic history as a result of his 3 gold medals in Helsinki in 1952

Kuts was renowned for surging bursts of speed in every lap as a means of intimidating his opponents, the very opposite of the theoretically more efficient even pace running.

Pirie’s figures are notable for the fastest first kilometre until almost 20 years later, again raising questions of pace control.

Continued....

Clarke with his two records achieved the greatest reduction of the record by a single athlete ie 15.4 s altogether.

Moorcroft's record is the first to utilise 1/100 ths of a second, and Aouita the first to break it by 1/100 ths of a second; a ridiculously inappropriate measure over this distance, as the track expands and contracts by greater than that amount, and is not measurable in terms of these limits of accuracy. However, it does provide athletes and promoters with greater possibilities of world record runs.

Aouita was subsequently the first under the 13 minute 'barrier'.

Keino *(Kenya)* 13:24.2 in 1965 *(not listed as only 440 yard lap times available)* was the first African athlete to break this record, a sign of the changing times bringing wider opportunities for participation which were to lead to their present day dominance.

The earliest record for a Kenyan track athlete competing outside of East Africa is in 1952 in Malaya where Kipsong won the 5000 m in 15:03.4, while serving with the King's African Rifles. The first International appearance by a Kenyan team was in 1954 in London, whilst en route for the Commonwealth Games in Vancouver. Their first gold medal in International competition came in the 1962 Commonwealth Games in Perth in Australia, but in the 100 and 220 yards sprints! Their first Olympic medal was won by Kiprugut in the 800 m at the 1964 Tokyo Olympic Games, who subsequently won the gold medal in the same event at the 1968 Mexico Olympic Games.

With a population of about 70 million, about half of which are under 15 years old, Kenya has produced a disproportionate number of internationally ranked track runners. In 1995 Kenya had 77 athletes listed in the Top 50 rankings in the men's middle and long distance events *(800 m to Marathon)* compared to Britain's 15, giving a ratio of one listed middle and long distance track athlete to every 310 169 of the population, as opposed to Britain's one to every 3 804 333, and the USA's one to every 8 090 929.

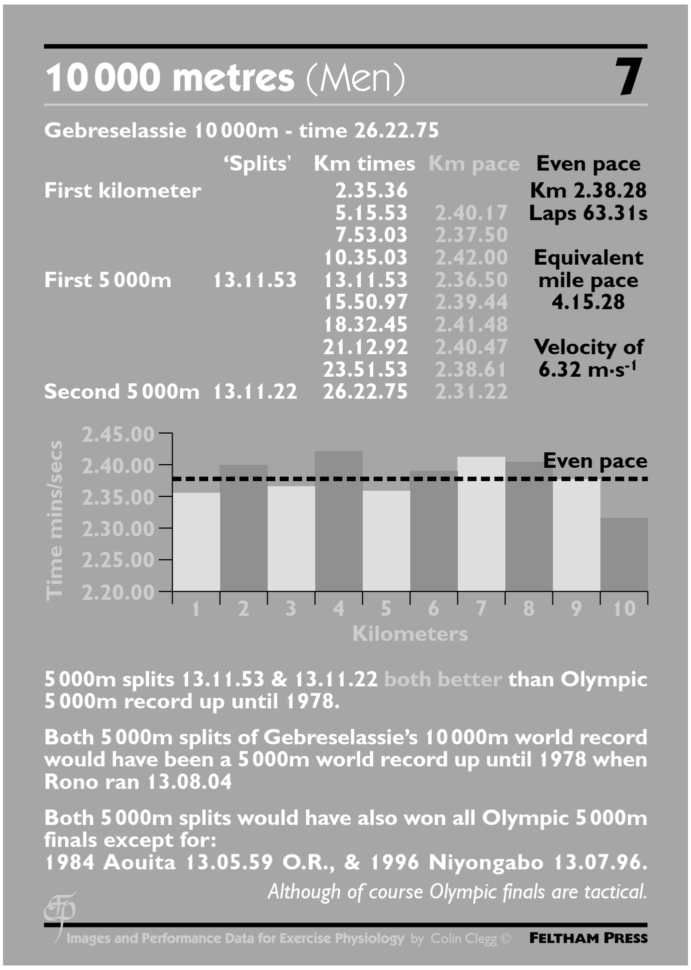
At the 1988 World Cross Country Championships, Kenyan runners filled 8 of the first 10 places, with the other 2 being Ethiopians. At the 1988 Olympic Games in Seoul, Kenyan runners won the 800 m, 1500 m, 3000 m Steeplechase, and the 5000 m. On the basis of population percentages, the probability of this happening by chance was estimated to be about 1 in 1.6 thousand million. As the top Kenyan runners come from tribes that constitute less than 20% of Kenya's population, the odds are a great deal greater than even this astronomical number.

Attempts to elucidate the underlying physiological explanation for these astonishing figures have been controversial and inconclusive. The most well known of these studies by Saltin 1995, was facilitated in Kenya by Keino who won the gold medal in the 1500 m at the 1968 Mexico Olympic Games.

Gebresellasie's *(Ethiopia)* performances are astonishing. The average of his 10 fastest races at this distance up to January 1996 was 13:02.82

10 000 metres *(Men)*

Image 7



10 000 metres (Men) **7**

Gebreselassie 10 000m - time 26.22.75

	'Splits'	Km times	Km pace
First kilometer		2.35.36	
		5.15.53	2.40.17
		7.53.03	2.37.50
		10.35.03	2.42.00
First 5 000m	13.11.53	13.11.53	2.36.50
		15.50.97	2.39.44
		18.32.45	2.41.48
		21.12.92	2.40.47
		23.51.53	2.38.61
Second 5 000m	13.11.22	26.22.75	2.31.22

Even pace
Km 2.38.28
Laps 63.31s

Equivalent mile pace 4.15.28

Velocity of $6.32\ m\cdot s^{-1}$

5 000m splits 13.11.53 & 13.11.22 both better **than Olympic 5 000m record up until 1978.**

Both 5 000m splits of Gebreselassie's 10 000m world record would have been a 5 000m world record up until 1978 when Rono ran 13.08.04

Both 5 000m splits would have also won all Olympic 5 000m finals except for:
1984 Aouita 13.05.59 O.R., & 1996 Niyongabo 13.07.96.

Although of course Olympic finals are tactical.

Images and Performance Data for Exercise Physiology by Colin Clegg © **FELTHAM PRESS**

When run at even pace, about 97% of the energy demands of this event are supplied aerobically. Tactical demands could increase the anaerobic component, as can psychological motivation when the end is in sight, and an all or nothing flat out finish can be attempted. This is clearly seen over the last kilometre in Gebreselassie's run, which was run at close to 4 minute mile pace *(60.5 s per lap)*.

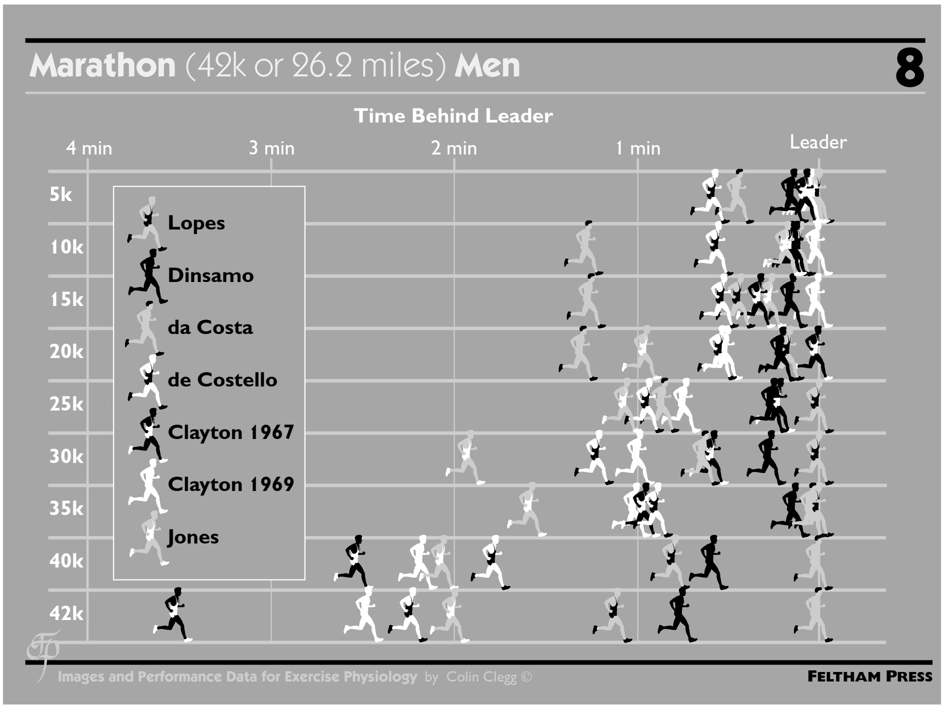
The average pace of this performance is such that each 200 m was run in 31.75s. For those not familiar with this pace, it could be of practical interest to attempt a 200 m at this pace, or even a 100 m in 15.9s.

As can be seen this race was run at close to even pace, with a maximum variation of 2.4% over the first 9 kilometres.

Considering the split times at 5000 m, then it is remarkably even pace, the difference between the two of 0.31 s representing a distance of just under 2 metres!

Marathon *(42 k or 26.2 miles)* **Men**

Image 8



Strictly, Marathons should be run on out and back courses in order to avoid any overall gain or loss in height of terrain, wind advantage, etc. This is not always the case, and athletes search out 'fast' courses, which are inevitably flat. As the courses vary so much, Marathon 'bests' rather than records are referred to.

When reviewing the data on the Marathon, the remarkably slow rate of improvement when compared to other events becomes apparent *(eg since 1967, there has been an improvement of only 2 mins:28.6 s in the Marathon best, a 2.7% improvement compared to 4.6% in the 5000 m).*

Many fewer Marathons are run than track events of course, which partially explains the relatively few 'bests'. However, the relatively slow rate of improvement also raises questions about the underlying physiological reasons, especially as considering the length of the Marathon it might be the event where most progress would be expected to be made; especially in the fields of nutrition and hydration.

All great Marathon runners are, or were, also top 10 000 m runners, and advances might be made towards the 2 hour 'barrier' if today's 10 000 m runners turn their attention to the distance whilst at their peak. For example using the 10 km to Marathon conversion constant of 4.667, Gebresellasie would have a projected time of about 2 hours 3 minutes 6 seconds for the Marathon.

That Marathon times have not improved dramatically in an era of the availability of widespread doping procedures, would tend to suggest that the limiting factor

Continued....

for performance in the Marathon is not to be found in either the oxygen supply pathways or in muscle development. An educated guess would suggest that the limits to performance in the Marathon are concerned with energy supplies, and the inescapable fact that “fats only burn in the flames of carbohydrates”. Thus as the intensity of the pace increases, and the glycogen stores are depleted, any attempts at maintaining blood glucose levels by drinking ‘energy drinks’ during the event have limited success due to the problems of passage through the stomach and absorption whilst running, and so the virtually limitless supplies of energy in the fats of the various compartments of the body cannot be fully exploited. It is the depletion of the glycogen stores which results in the sudden loss of pace, known as “hitting the wall”. Hence the efforts to increase glycogen stores by ‘glycogen loading’, in an attempt to avoid depletion of reserves during the race. It is suggested that physiologically speaking the 32 km *(20 miles)* mark is about “half way” in a Marathon, a ludicrous idea to anybody who has not attempted the distance, but it is past that point that the limiting physiological problems begin to be felt most dramatically.

Interestingly with regard to pace control and ‘glycogen sparing’ in the early part of the race, da Costa was second slowest to 5 k, the slowest to 10 k, 15 k, and 20 k, and second slowest to 25 k; and was only ‘ahead’ at 40 k.

The slowest 5 k was that of Clayton’s 35 k - 40 k section which was run at 5:09 mile pace, and the fastest was da Costa’s 35 k - 40 k section which was run at 4:38 mile pace. The overall speed of da Costa being 12.5 mph.

The Women’s Marathon is a fairly recent event, with restricted opportunities, and there is no evidence yet of a dramatic closing of the differential with men that was predicted in the 70’s and 80’s, and might be expected from physiological considerations. The 2 hr 20 mins ‘barrier’ was broken by Jim Peters in 1953, but has still *(1998)* to be bettered by a woman.

The first Marathon to permit women entries was the Isle of Wight Marathon in 1964, when Dale Greig *(GBR)* started 4 minutes ahead of a field of 67 men, and finished in 3:27:45.

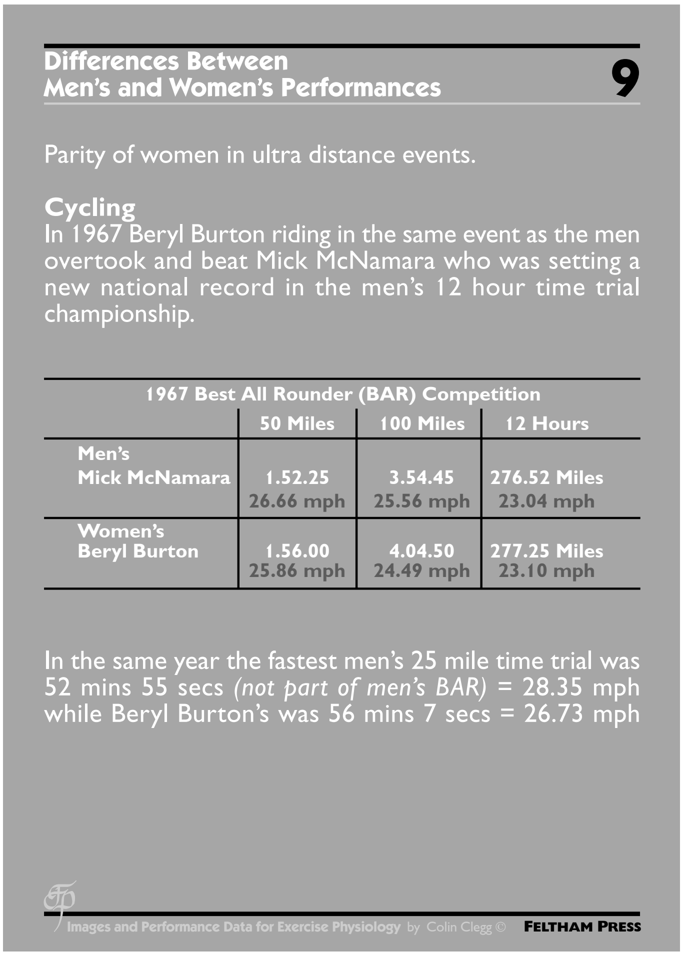
The first Women’s Only Marathon to be run in Britain was run in **Feltham** in 1976, being won by Christine Readdy in 2:50:55

The first woman under 3 hours in an officially recognised event was Elizabeth Bonner *(USA)* who ran 2:55:22 in New York in 1971, finishing more than half an hour behind the first man.

The first woman under 2 hours 30 mins was Grete Waitz *(NOR)* who finished in 2:25:28.7 in the New York Marathon for Women in 1980.

Differences between Men's and Women's Performances

Image 9



Differences Between Men's and Women's Performances

9

Parity of women in ultra distance events.

Cycling
In 1967 Beryl Burton riding in the same event as the men overtook and beat Mick McNamara who was setting a new national record in the men's 12 hour time trial championship.

1967 Best All Rounder (BAR) Competition

	50 Miles	100 Miles	12 Hours
Men's Mick McNamara	1.52.25 26.66 mph	3.54.45 25.56 mph	276.52 Miles 23.04 mph
Women's Beryl Burton	1.56.00 25.86 mph	4.04.50 24.49 mph	277.25 Miles 23.10 mph

In the same year the fastest men's 25 mile time trial was 52 mins 55 secs *(not part of men's BAR)* = 28.35 mph while Beryl Burton's was 56 mins 7 secs = 26.73 mph

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

It is now widely accepted that previous contentions that the gap between male and female performances would narrow until parity was reached were unrealistic, being based on projections or extrapolations from the rising level of female performance, as a result of increased participation/expectation/ and some cases drug abuse. For example projections in 1992 on the male and female marathon times met at 1998, whereas in reality at that time the men's time for the marathon was 2 hours 6 minutes 5 seconds *(Ronaldo da Costa of Brazil)*, and for the women's 2 hours 20 minutes 47 seconds *(Tegla Laroupe of Kenya)*.

This situation prompts a consideration of the differences between projections and predictions. A projection from present data is not a prediction. Such considerations are also involved when dealing with the progress of performance of individuals. A very small change in slope of a curve can lead to large changes in projections, depending upon the length of time over which it is extended. The longer the time the greater the divergence.

Continued...

Now that the improvement rate of women's performances has levelled off, sex differences from the mile to the marathon have stabilised, so obviously future projections based on these are greatly revised suggesting a slowing of the closing of the gap.

The problems of opportunity and participation arise with all analyses of women's performances, for example in the 1980 Olympic Games there were 1088 women competitors, and in the 1996 Olympic Games there were 3779 women competitors, an increase of 350%. The first female athlete to win an Olympic Gold medal for a track event was Betty Robinson *(1911-1999)* at the 1928 Games in Amsterdam when she was 16 years of age.

In some cases, such as ultra endurance events, the performance of women can be on a par with those of men. A good example of this being seen in the late great Beryl Burton *(1937-1996)*, perhaps the greatest woman endurance athlete of all time, who beat the men's British record for the 12 hour time trial in the same event as Mick McNamara was setting a new British Record in 1967. A truly remarkable occurrence in the annals of endurance sport.

With regard to the gap between men and women in the speed endurance and strength events, it is significant that the widest used group of doping substances are anabolic steroids which are all derivatives of the male hormone testosterone, no sportsperson uses the female hormones to improve strength and endurance.

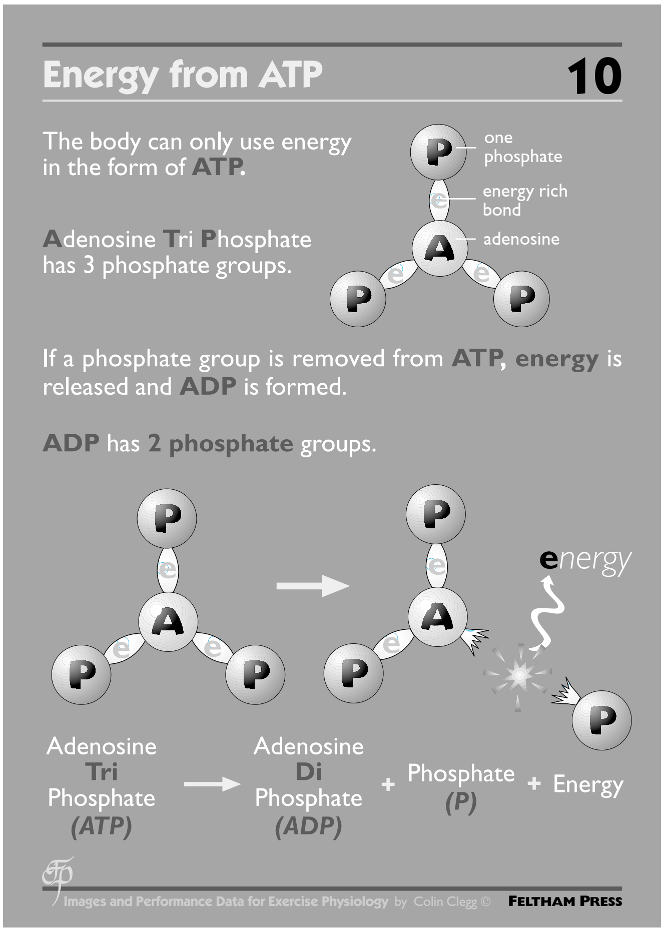
However, oestrogen does have anabolic effects, particularly in females, which is why it is widely used as an animal feed additive, but its main effect on the muscles appears to be a role in protection from muscle damage as a result of exercise. Although it has been noted that hormone replacement therapy *(HRT)* in post-menopausal women prevents excessive loss of muscle force production. *(It has been reported that female gymnasts have become pregnant and aborted, in an attempt to manipulate hormone levels in a way that promotes flexibility and recovery.).*

Oestrogen also has a role in fat metabolism, which may favour fat use as an energy source over longer distances, although the basic dependence on the breakdown of glycogen remains the same in males and females.

Although the mechanisms are not fully understood, women tend to slow down less than men with increasing race distance or time passed. It has been asserted that generally, with a group of men and women with the same 10 km time, the women will perform better than those men over the Marathon distance, and even more so over distances greater than the Marathon, most likely as a result of differences in long term fat metabolism.

Energy from ATP

Image 10



Energy from ATP **10**

The body can only use energy in the form of **ATP**.

Adenosine **T**ri **P**hosphate has 3 phosphate groups.

If a phosphate group is removed from **ATP**, **energy** is released and **ADP** is formed.

ADP has **2 phosphate** groups.

Adenosine **Tri** Phosphate *(ATP)* → Adenosine **Di** Phosphate *(ADP)* + Phosphate *(P)* + Energy

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

The body can only use energy in the form of ATP. ATP is an abbreviation for adenosine triphosphate. ATP is referred to as an ‘energy rich’ molecule.

It is essential to provide some idea of the scale of the molecular world for an intelligent appreciation of the subject. 484g of ATP *(about the weight of a typical tin of baked beans)* contains about 6 x 10^{23} molecules or 600 thousand million million million, a number that would take about 20 thousand million million years to count non-stop *(if you had the time)*.

Although in total the average person contains only about 50 g of ATP *(the weight of about 3 cheese triangles)*, because this ATP is continuously recycled in the body, being broken down and resynthesised all the time, the body uses a massive amount over a 24 hour period. For example a moderately active person would use or ‘turn-over’ a weight at least equivalent to their body weight in a 24 hour period, and this figure would be many times higher in an active athlete.

Continued....

ATP is usually described as losing only one phosphate group when yielding energy, but under increasing demand when exercise intensity is high and the rate at which ATP is broken down exceeds its resynthesis, ADP accumulates and can yield further energy in being converted to AMP.

$$\text{ATP} \rightarrow \text{ADP} + \text{P} \rightarrow \text{AMP} + \text{P} + \text{P}$$

$$\text{ADP} + \text{ADP} \rightarrow \text{ATP} + \text{AMP}$$

To avoid confusion with ‘correct’ chemistry, it must be realised that the description of energy being released when an ‘energy rich’ bond is broken, and being trapped when an ‘energy rich’ bond is formed, is only a model. Srictly speaking energy is required to break chemical bonds, and is released on their formation. ATP is involved in energy transferring reactions.

It is also important to remember that any energy not trapped in chemical bonds is released as heat and is not available for useful mechanical or chemical work.

At this level it is best to avoid discussions about the absolute number of ATP molecules that are obtained per molecule of fully oxidised glucose, fat and/or protein. The biochemistry required is beyond the needs of most students of PE and Sport Studies, and the literature contains differing estimates. The different figures seen are based on different models of mechanisms for the transfer of energy from the respiratory substrate into ATP molecules

Without a thorough understanding of chemistry, detailed descriptions of those pathways, as often given in P.E. texts at this level, are little more than lists of names, and add little or nothing to the reader’s understanding, often causing anxiety and confusion amongst candidates.

This is particularly true of detailed calculations of the number of molecules of ATP generated from the oxidation of each molecule of substrate via these pathways.

Much time is frequently spent at this level in demonstrating that 38 molecules of ATP are produced from one molecule of glucose via the Krebs cycle and electron transport chains in aerobic respiration in the mitochondria.

However, amongst specialists in the field of bioenergetics *(the ‘mitochondriacs’)* the consensus of opinion is that aerobic oxidation of one molecule of glucose yields 31 molecules of ATP. The emphasis being on the number of protons *(H^+)* needed for ATP synthesis, and the number of protons pumped across the inner mitochondrial membrane, according to Mitchell’s Chemiosmotic Theory, rather than on the flow of electrons down the cytochrome electron transport chains.

The same argument applies to the yield of ATP molecules from the oxidation of a molecule of palmitate *(hexadecanoate)* the most abundant fatty acid in the body, where the ‘traditional’ figure is 129 and the ‘mitochondriacs’ figure is 102.

(Reference: Brand M. D. The Biochemist, August 1994, 20-224)

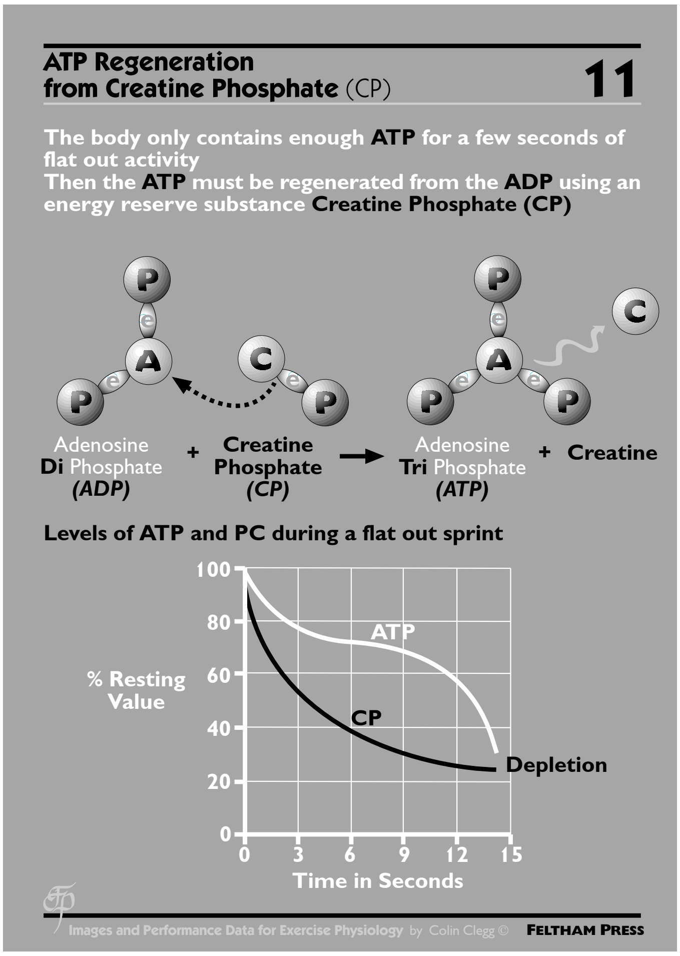
Continued....

The essential point is that the anaerobic glycolysis of a molecule of glucose yields relatively small amounts of ATP rapidly, and that the complete oxidation of a molecule of glucose via aerobic pathways in the mitochondria yields considerably larger amounts *(approximately x 15)* of ATP less rapidly, and that of fatty acids even larger amounts *(approximately x 50)* albeit at a higher O_2 cost than the oxidation of glucose.

Efficiency of respiration = energy trapped as ATP/ Total energy released x 100.

ATP Regeneration from Creatine Phosphate *(CP) (Also known as Phospho-creatine or PCr)*

Image 11



ATP Regeneration from Creatine Phosphate (CP) **11**

The body only contains enough **ATP** for a few seconds of flat out activity
Then the **ATP** must be regenerated from the **ADP** using an energy reserve substance **Creatine Phosphate (CP)**

P
e
A
P
e
C
e
P
P
e
A
P
e
e
P
C
Adenosine **Di** Phosphate *(ADP)*
+
Creatine Phosphate *(CP)*
Adenosine **Tri** Phosphate *(ATP)*
+
Creatine

Levels of ATP and PC during a flat out sprint

100
80
60
40
20
0
% Resting Value
ATP
CP
Depletion
0
3
6
9
12
15
Time in Seconds

Images and Performance Data for Exercise Physiology by Colin Clegg © **FELTHAM PRESS**

An estimate of the amount of ATP present in the whole resting body is on average about 50g.

Any activity increases the rate of energy use via ATP, and it must be regenerated from other sources. The most immediate source being creatine phosphate *(or phosphocreatine)*. ATP & CP being collectively known as the alactic anaerobic phosphagen system.

The graph shown is an approximation only, and the horizontal scale might be 'reduced' to between 1-6 seconds by some authorities. Also it must be remembered that all energy supply systems operate at all times, although the anaerobic lactic system would show the most immediate increase in maximal efforts.

The amount of ATP in the body appears to be relatively fixed, but the amount of creatine phosphate can be increased significantly by training, and by a dietary supplementation regime of creatine.

Continued...

Normally there are approximately 120g of creatine in the body, 95% of which is found in the muscles. As well as being part of the diet, creatine is also synthesised by the body in the liver and the pancreas.

The involvement of creatine in muscle metabolism was first postulated in 1847 when Lieberg reported that the muscles of wild foxes contained about ten times more creatine than that of captive and hence more sedentary animals. A situation which will be mirrored in a comparison of active and non-active humans!

In addition to its role in the resynthesis of ATP from ADP, creatine has been shown to stimulate the synthesis of muscle protein, so that creatine supplementation results in increased performance during high-intensity intermittent strength/power training.

Dietary supplementation with creatine is a contentious subject, at present *(1999)* not banned, and widely used even down to club level. It is accepted on the grounds that it is a normal component of the diet, especially in meat eaters, and does not alter the nature of the body as such. Human muscle has an upper limit of creatine content which cannot be exceeded.

Excess creatine is broken down and excreted by the liver and kidneys.

Homeostasis, with its negative feedback control, ensures that supplementation with creatine suppresses its synthesis by the body, which returns to normal levels when supplementation ceases. This phenomenum of homeostatic negative feedback control is seen in all cases where supplementation is used to raise levels of substances in the body, for example the use of anabolic steroids *(testosterone derivatives)* results in the natural synthesis of testosterone being suppressed.

The basic regime of creatine supplementation is 20g/day for 5-6 days; with a maintenance dose of 2 - 5g per day, the latter being about the same as would be present in an average meat eater's diet.

Reservations on its use have as yet *(1999)* no firm foundation. Creatine supplementation causes an increase in creatinine in the urine, which is often used as an indicator in the study of kidney function, but this is not a sign of abnormal kidney function in these cases, but simply reflects the greater turnover of creatine in those performing supplementation.

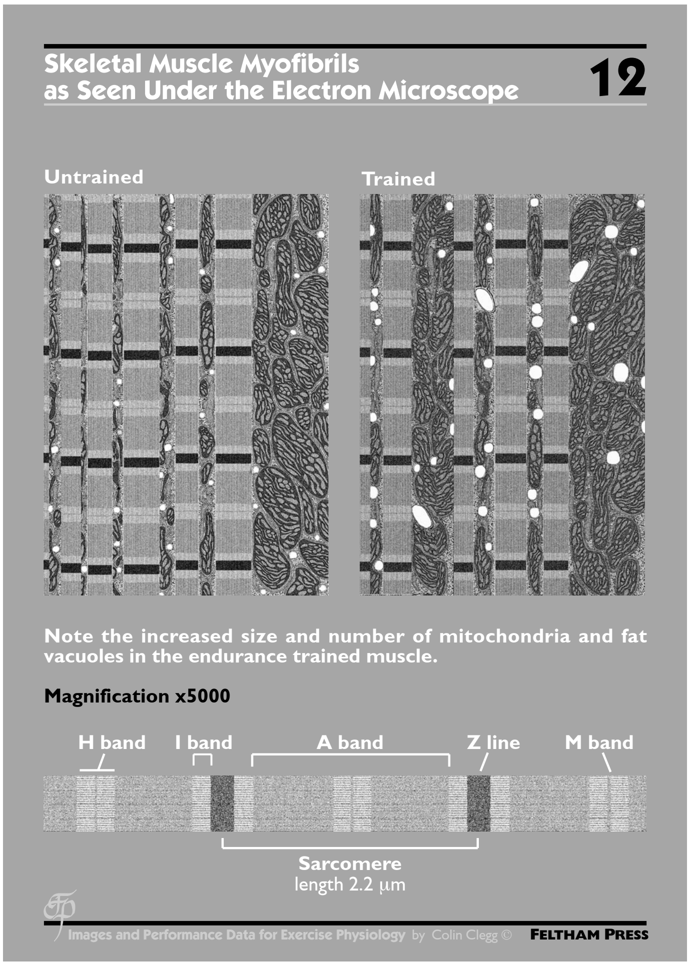
Creatine supplementation has been compared to that other accepted dietary regime of carbohydrate or glycogen 'loading', which is widely practised amongst many sportspeople.

It is claimed that creatine supplementation is one of the most significant developments in sports related nutrition since the development of carbohydrate or glycogen loading in the 1960's.

Creatine supplementation is in fact more effective when undertaken in combination with carbohydrate supplementation, and with sodium phosphate which also appears to increase levels of 2,3-DPG in the red blood cells, which promotes oxygen release to the tissues.

Skeletal Muscle Myofibrils

Image 12



Skeletal Muscle Myofibrils as Seen Under the Electron Microscope **12**

Note the increased size and number of mitochondria and fat vacuoles in the endurance trained muscle.

Magnification x5000

Sarcomere length 2.2 µm

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

Longitudinal section seen under the electron microscope

There should be a spontaneous interest in muscle contraction, as it is such an obvious part of our everyday lives, and the gross effects are so easily observed.

However, the relationship between these everyday observations and the underlying microscopic and molecular events can present problems.

This gap can best be bridged by paying careful attention to matters of scale and size. A clear idea of these should render descriptions of the sliding filament theory more relevant to students.

If raw meat *(ie muscle)* is teased out in water on a microscope slide, and the light correctly adjusted, then very fine cross striations can be seen under low power *(×100)* magnification. These can be seen more clearly on pre-stained slides, but here the immediate relation to real muscle is lost. No extra detail is revealed under high power *(×400)* as the amount of detail revealed *(the resolution or*

Continued....

resolving power) is limited by the wavelength of light - greater and greater magnification reveals no extra detail, rather like endlessly magnifying a picture in a newspaper.

For example mitochondria can just about be seen under the light microscope as tiny specks when stained with special stains. Further detail can only be revealed by use of the electron microscope, which has a resolving power about X200 greater than that of the light microscope, and magnifies up to X500 000 *(a power of magnification equivalent to enlarging the tip of a ball point pen to a sphere of about 500 metres in diameter).*

However, it must be remembered that, despite its limitations, the original elucidation of the sliding filament mechanism was achieved as a result of observations under the light microscope. In fact all the observations that had led to the sliding filament idea had been published in Germany between 1858 and 1876, but subsequently neglected until the 1950's. Similarly light microscope work in 1902 had indicated the presence of the endoplasmic reticulum. This incredible work by the light microscopists has all subsequently been confirmed and enlarged upon by work with the electron microscope.

The electron microscope does not use light, but makes use of a stream of electrons which are focused by magnets onto a fluorescent screen, of which photographs like the one shown are taken. Therefore there is never any true colour in an electronmicrograph as no light is involved. Although they are sometimes coloured artificially, this practice seriously undermines the fundamental concept of size and resolution; as even students with little understanding of resolution and magnification could not fail to be impressed by the fact that these objects are too small to be 'hit' by light.

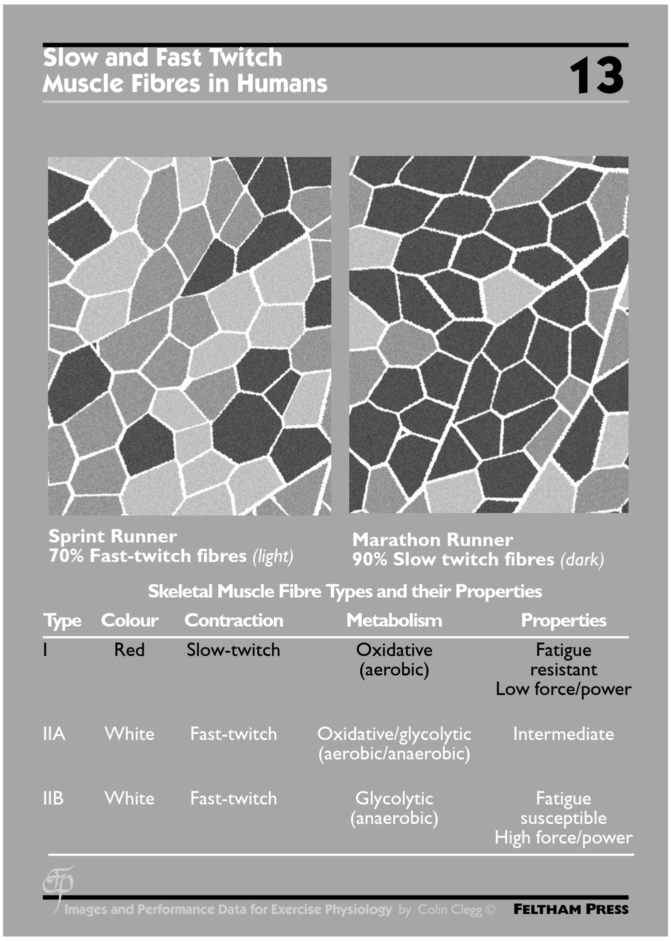
To further reinforce understanding of size and scale, various calculations could be made from the average diameter of a mitochondrion, which is about 1.0 micrometre, i.e. 1/1000 mm. In other words about 1000 mitochondria would fit into the diameter of the tip of a ball point pen.

Such considerations of size and scale are essential for an appreciation of the ultrastructure of muscles, and thus the sliding filament mechanism.

The banding pattern of striated muscle myofibrils and fibres, and the spatial arrangement of the mitochondria alongside the myofibrils clearly illustrates the relationship of structure to function.

Slow and Fast Twitch Muscle fibres in humans

Image 13



Slow and Fast Twitch Muscle Fibres in Humans **13**

Sprint Runner
70% Fast-twitch fibres *(light)*

Marathon Runner
90% Slow twitch fibres *(dark)*

Skeletal Muscle Fibre Types and their Properties

Type	Colour	Contraction	Metabolism	Properties
I	Red	Slow-twitch	Oxidative (aerobic)	Fatigue resistant Low force/power
IIA	White	Fast-twitch	Oxidative/glycolytic (aerobic/anaerobic)	Intermediate
IIB	White	Fast-twitch	Glycolytic (anaerobic)	Fatigue susceptible High force/power

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

Skeletal muscles in humans contain mixtures of Slow Twitch Type I *(red)* fibres and Fast Twitch Type II *(white)* fibres; with the Type II fibres being further differentiated into IIA Fast Oxidative-Glycolytic *(FOG)* and IIB Fast Glycolytic *(FG)*.

A predominance of slow fibres will favour aerobic endurance activities. They are longer and thinner than fast fibres and result in relatively slender muscles which are very difficult to increase in bulk.

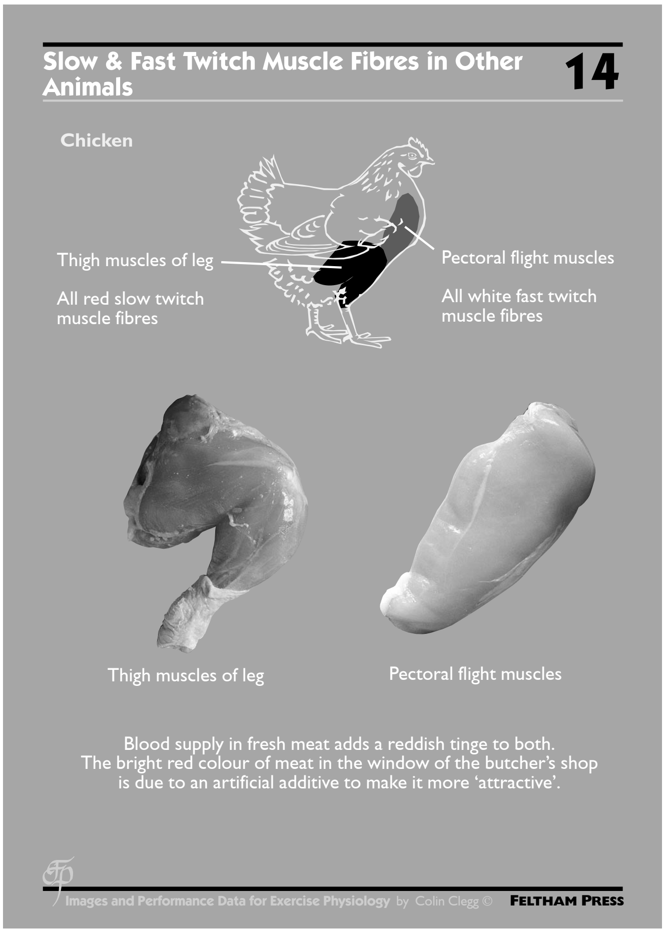
A predominance of fast fibres will favour anaerobic power activities and result in relatively bulkier muscles which are more easily increased in mass. IIA fibres have a mixture of aerobic *(oxidative)* and anaerobic *(glycolytic)* properties, and IIB fibres are purely anaerobic.

The particular proportion of each type of fibre in a muscle is determined genetically, but their properties can be changed by training, with for example Type IIA fibres in endurance trained individuals being more 'aerobic' than Type I fibres in untrained individuals.

Also the the presence of a third undifferentiated Type IIC fibre reported by some, may provide some scope for an apparent change in proportion of fibres with training.

Direct observation of Slow and Fast Twitch Muscle Fibres in Other Animals

Image 14



Slow & Fast Twitch Muscle Fibres in Other Animals **14**

Chicken

Thigh muscles of leg

All red slow twitch muscle fibres

Pectoral flight muscles

All white fast twitch muscle fibres

Thigh muscles of leg

Pectoral flight muscles

Blood supply in fresh meat adds a reddish tinge to both.
The bright red colour of meat in the window of the butcher's shop
is due to an artificial additive to make it more 'attractive'.

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

Other animals have a more distinct division of fast and slow type skeletal muscles, and direct observation of such 'meat' can provide interesting illustration of the observable differences between slow and fast twitch muscle fibres, whilst relating the theory very closely to everday life.

Chicken are recent descendants of jungle fowl still to be found in the SE Asian jungles, adapted for long 'aerobic endurance' hours of scratching about in the leaf litter of tropical rainforests, whilst being capable of very rapid powerful 'anaerobic' bursts of near vertical take off to the tops of trees to escape predators, and when roosting at night.

Continued....

These capabilities are reflected in the well known characteristics of chicken 'meat'. The leg muscles are composed of Type I aerobic endurance 'red' fibres and the meat is 'red', and the flight *(breast)* muscles are composed of Type II anaerobic 'white' fibres and the meat is 'white'.

These characteristics and abilities remain in most domesticated chicken despite years of selective breeding for egg and/or meat production.

Muscle fibres in humans can be up to several centimetres long, and can be branched, but do not usually run the entire length of an anatomical muscle. However, for meat eaters there is an interesting observation to be made about the tenderness of 'fillet beef steak', which is prepared from the psoas muscle. In this muscle the muscle fibres do run the entire length of the anatomical muscle, thus reducing the amount of tough connective tissue needed to bind them together, which results in a more tender meat.

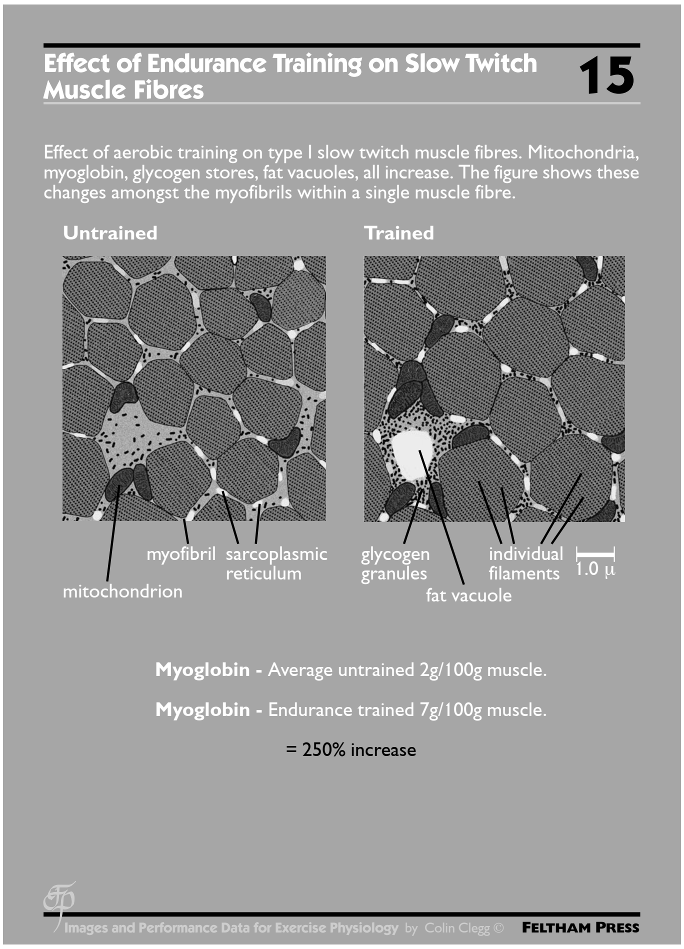
Observations by Lieberg in 1847 that the muscles of wild foxes contained about ten times more creatine than that of captive and hence more sedentary animals led to the idea of the involvement of creatine in muscle metabolism.

The thin layer of red 'meat' seen just under the skin of fish such as the mackerel is in fact a layer of slow Type I 'red' fibres used in long slow 'endurance' swimming. This muscle is 'red' as a result of the high levels of myoglobin, mitochondria, and blood capillaries.

The bulky blocks of typical white fish 'meat' making up the main mass of the body are blocks of fast Type II 'white' fibres used in explosive escape or attack movements. This muscle is 'white' due to the absence of myoglobin, mitochondria, and blood capillaries.

Effect of Endurance Training on Slow Twitch Muscle Fibres

Image 15



Effect of Endurance Training on Slow Twitch Muscle Fibres **15**

Effect of aerobic training on type I slow twitch muscle fibres. Mitochondria, myoglobin, glycogen stores, fat vacuoles, all increase. The figure shows these changes amongst the myofibrils within a single muscle fibre.

Myoglobin - Average untrained 2g/100g muscle.

Myoglobin - Endurance trained 7g/100g muscle.

= 250% increase

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

As seen in cross section under the electron microscope

Although the adaptations of muscles to aerobic endurance work show the most dramatic changes, some points of interest can be noted with regard to strength training.

There is only a weak correlation obtained between human muscle fibre composition and muscle force.

Absolute strength may be more dependent upon the size of the individual fibres irrespective of type, and the number of fibres per motor unit - not upon differences in force produced by each fibre type, eg there are many more fibres per fast twitch motor unit than slow twitch motor unit. Maximum effort achieves full motor unit activation.

Continued...

Maximum strength is proportional to the number of active cross bridges, cycle rate and duration of attachment of the cross bridges, overall muscle mass, fibre length *(fast twitch are shorter than slow twitch)*, fibre angle, functional cross sectional area of the muscle i.e. the cross-sectional area of muscle fibres *(which is different from the anatomical cross sectional area, which would include fat)*, motor unit activation, and many other factors.

Early strength gains in resistance training are mainly as a result of neural adaptation resulting in increased activation and coordination. The fact that artificial stimulation of a de-ennervated muscle will not lead to hypertrophy, and in fact cannot even prevent atrophy, indicates that the nerve cell supplying the motor unit also has a trophic function essential for protein synthesis within the muscle fibre.

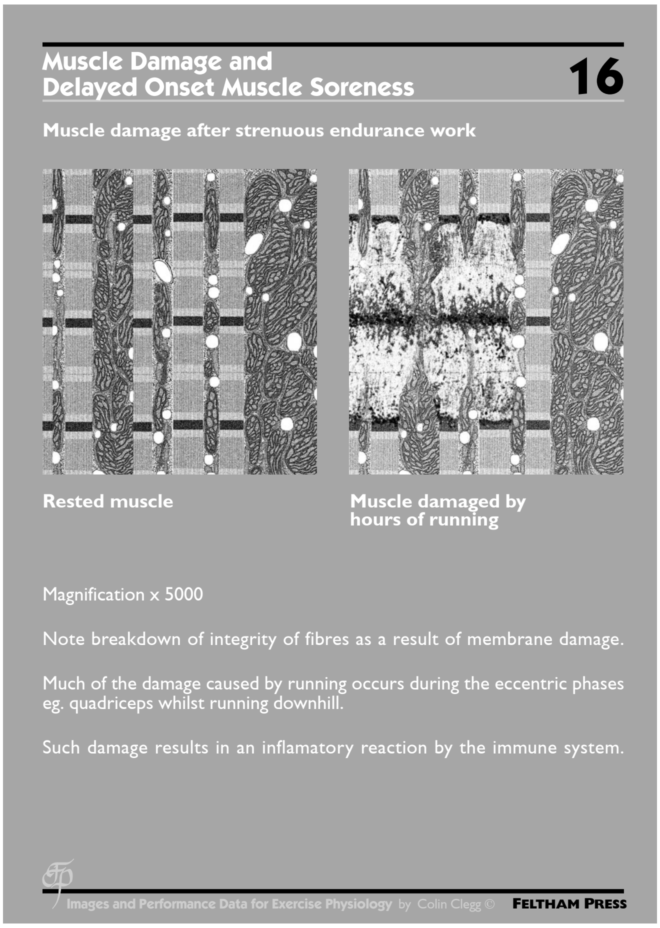
Training programmes to increase muscle size and strength remain a matter of personal choice, and results are not predictable within the limits of statistical certainty. As with all responses of the body to demand there is a large inherited component in the development of lean body mass ie muscle development. General observation will reveal ‘lazy’ athletes who increase muscle mass easily, and hard training athletes who despite being very strong hardly make any gains in muscle mass at all. What is interesting to note is the almost universal belief that musclature, and hence body shape, can be ‘sculpted’ almost to order, despite observations to the contrary especially if one attends a ‘fitness suite’ at health centre. Even the role of exercise in weight *(fat)* reduction is grossly over-estimated among the general population. Unfortunately, the body is super efficient in its energy economy. However, should a regular exerciser manage to lose weight with a combination of exercise and a reduction in food intake *(mainly by a reduction of fats)* any improvement in musclature observed, could be more to do with the reduction in sub-cutaneous fat revealing the underlying musclature that was already there, than with dramatic muscle hypertrophy.

The arguments about the interconvertibility of the different types of muscle fibres remains *(1999)* unresolved. The safest position to take at the moment is that they are not interconvertible under normal conditions, the only widely accepted example of interconversion being the result of switching motor nerves between fast and slow twitch motor units. An observation reinforcing the fact referred to above, that the motor neurone has a complex relationship with the muscle fibres in a motor unit over and above the initiation of contraction via its nerve impulse.

The debate is confused by considerations of the nature of the so-called ‘intermediate muscle fibres’ which have been considered as a pool of ‘labile’ fibres capable of conversion one way or the other, and/or as representing fibres of either type in a transitional phase during their conversion from one type to another. Certainly if conversion was a major event as a result of strenuous training the problem would have been resolved by now.

Muscle Damage & Delayed Onset Muscle Soreness as seen under the electron microscope

Image 16



Muscle Damage and Delayed Onset Muscle Soreness **16**

Muscle damage after strenuous endurance work

Rested muscle

Muscle damaged by hours of running

Magnification x 5000

Note breakdown of integrity of fibres as a result of membrane damage.

Much of the damage caused by running occurs during the eccentric phases eg. quadriceps whilst running downhill.

Such damage results in an inflamatory reaction by the immune system.

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

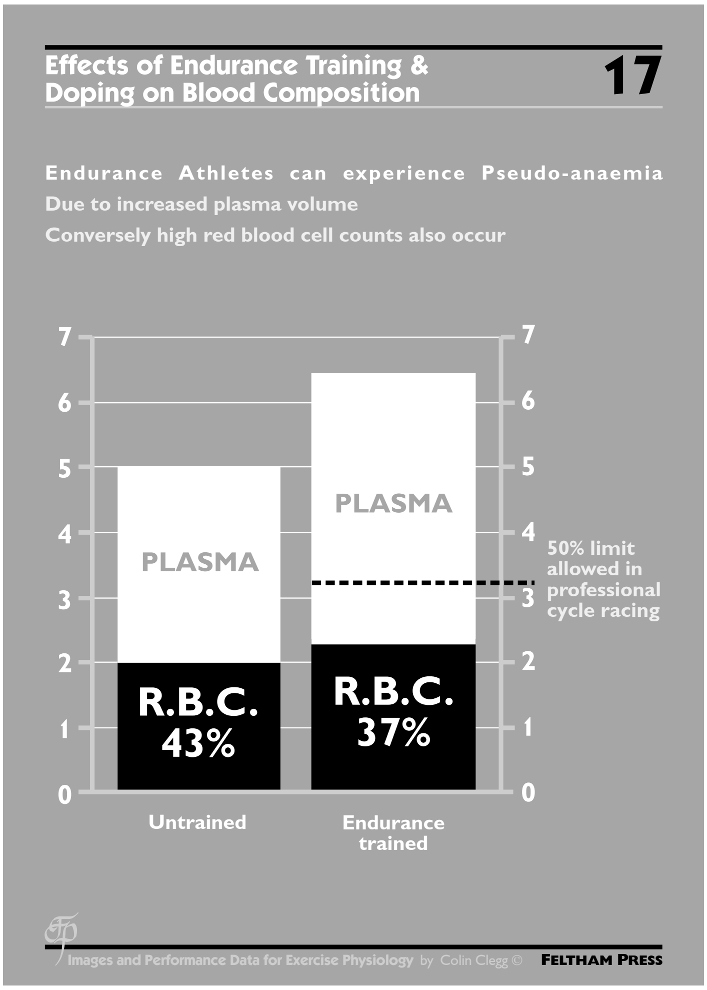
Unaccustomed and/or extreme exertion results in tissue damage, both of the connective tissue between the muscle fibres, and the fibres themselves.

Membranes are damaged, Z-discs are ruptured, fibres are torn, capillaries are broken, and many other disruptions to the delicate sliding filament mechanism occur. Inevitably these contribute to fatigue and to delayed onset muscle soreness *(DOMS)*, which is felt particularly around the junctions of muscles with tendons, where damage may be greatest but where there are also more pain receptors. Intracellular substances, including myoglobin and enzymes eg. creatine kinase *(CK)*, are released into the bloodstream, and used as ‘markers’ for the extent of muscle damage.

This cellular damage triggers an inflammatory response of the immune system, in which white cells move in to remove cell debris. In fact, initially, for a short time the immune system is suppressed, possibly to damp down the auto-immune response in the face of the immediate damage - it is suggested that during this period immediately after the event, sportspeople are particularly prone to infection. Given sufficient recovery time, and the correct nutrition, the damaged muscle tissue is rebuilt stronger than it was before the damage. The aim of designing a training programme that damages to an extent that can be successfully repaired is the principle behind all muscle training regimes.

Effects of endurance training and doping on blood composition.

Image 17



Effects of Endurance Training & Doping on Blood Composition 17

Endurance Athletes can experience Pseudo-anaemia

Due to increased plasma volume

Conversely high red blood cell counts also occur

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

One of the most significant endurance training effects is that of increased plasma volume. Although there is a simultaneous increase in the number of red blood cells *(RBC)*, this increase is less than that of the increase in plasma volume, so that the % RBC by volume of blood decreases, giving rise to the condition of endurance athlete's pseudoanaemia. The % RBC by volume is determined by centrifuging a sample of blood, and is also known as the packed cell volume or haematocrit, although alternative techniques are now available.

The misuse of the hormone erythropoietin or EPO has brought the study of blood composition to the forefront of exercise physiology. EPO is naturally produced by the kidneys, and is increases the synthesis of RBC by the red bone marrow, in response to oxygen stress, as for example in endurance training, particularly at altitude.

Continued....

EPO can now be produced commercially by genetic engineering, and was originally for medical use. The misuse of EPO by endurance athletes, especially cyclists, had been widely suspected, and matters came to a head in the 1998 Tour de France EPO scandal, in which many top cyclists and teams were implicated. There are many problems with regard to raised red blood cell production which are very difficult to unravel. The quest for a reliable test for the use of EPO has, to date *(1999)*, proved elusive.

Some people have naturally high RBC levels, for example one family with such an inherited high level won medals in endurance events in the Winter Olympics over several generations. Altitude training has been used for decades for the same purpose and is completely accepted. Although it must be noted that the effectiveness of altitude training is still far from agreed amongst the various authorities, and it can go badly wrong. The use of 'decompression hotels' in which the air pressure was reduced was an interesting variant on this theme of increasing red blood cell count as a result of prolonged exposure to a reduced partial pressure of oxygen. Another variant was the development of portable decompression tents in which athlete's could sleep. All these techniques have their effect by stimulating an increase in EPO production, which in turn stimulates the increased production of red blood cells by the red bone marrow.

Blood doping, in which a volume of blood is removed from the body and the RBC are reinfused once the body has made up the original deficit, has been superceded by the availability of EPO. Blood doping is also undetectable.

To cut through this maze, professional cycling has set an arbitrary figure for the haematocrit of 50%, which disqualifies a rider from all competition until it drops below that figure. The reason for an athlete having a level of 50% is at the moment *(1999)* not taken into consideration. Although much criticised, professional cycling does take blood samples from athletes at regular intervals, and publishes the findings.

Levels above 50% offset the increase in plasma volume that occurs as a result of endurance work, and result in a dangerous and sometimes lethal increase in the viscosity of the blood. The deaths of many young athletes in Europe have been suspected as being due to EPO abuse.

There is still much controversy about the figures for haematocrits. Figures quoted for the range of haematocrit values in the population vary eg. 42 - 54% *(Astrand)* and 40 - 45% *(Costill)*. Although figures can be quoted eg. trained at sea level - 48%, 6 weeks training at 4500 m - 59%, EPO use over a period of time - 60+%, figures for haematocrits as a result of various activities will be unique to a particular individual. Haematocrit values are not widely quoted for the African runners, nor indeed for endurance runners as a whole, in contrast to professional cycling.

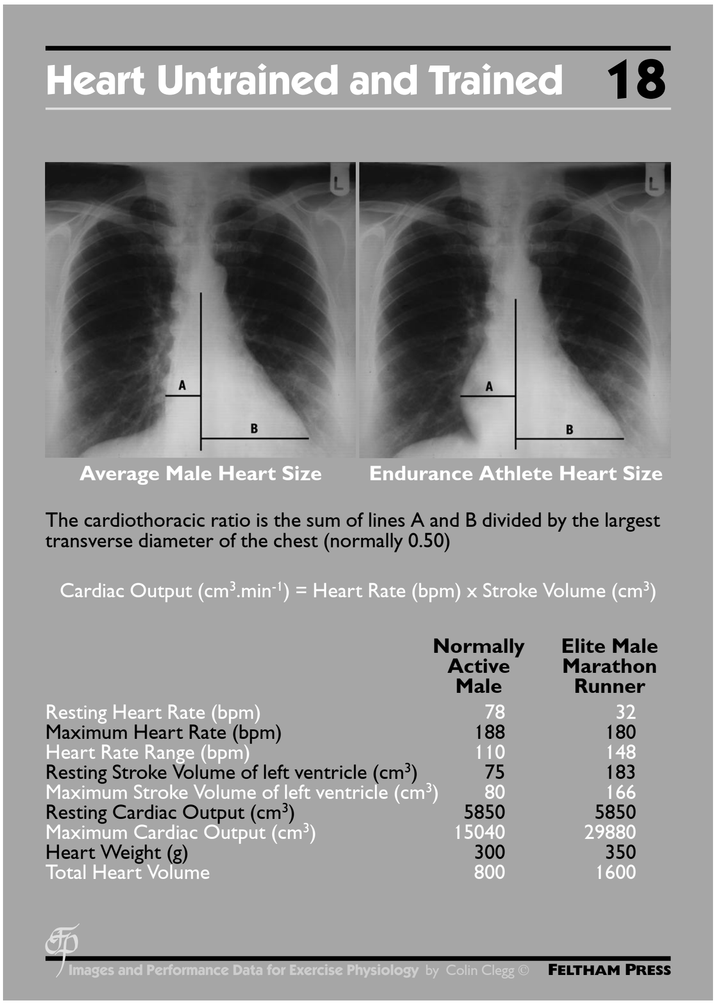
Continued....

The percentage of red blood cells by volume of blood relates directly to the amount of haemoglobin, which in turn determines the volume of oxygen that can be transported as oxyhaemoglobin to the tissues. The average haemoglobin content of the blood is 15.8 g per 100 cm^3 of blood in males *(range of 14 g - 18 g)*, and 13 .7 g per 100 cm^3 of blood in females *(range of 12 g - 16 g)*. The Federation International de Ski *(FIS)* bases its limits on these haemoglobin concentrations, rather than the haematocrit value, setting them at 18.5 g per 100 cm^3 of blood in males, and 16.5 g per 100 cm^3 of blood in females. At a roughly 3:1 relationship between haemoglobin concentration and haematocrit, these haemoglobin figures approximate to haematocrits of above 50% for males and just below 50% for females.

The short term effects of dehydration, diet, and posture on any of these blood measures, especially in ultra-endurance and multi-stage events, renders the standardisation of test procedures in the field difficult, and these difficulties fuel the controversy that still surrounds all attempts to tackle the problem of doping techniques relating to manipulation of the blood's oxygen carrying capacity.

Capacities of the 'normal' and 'athlete's' heart of subjects matched for size and age

Image 18



Heart Untrained and Trained 18

Average Male Heart Size **Endurance Athlete Heart Size**

The cardiothoracic ratio is the sum of lines A and B divided by the largest transverse diameter of the chest (normally 0.50)

Cardiac Output ($cm^3.min^{-1}$) = Heart Rate (bpm) x Stroke Volume (cm^3)

	Normally Active Male	Elite Male Marathon Runner
Resting Heart Rate (bpm)	78	32
Maximum Heart Rate (bpm)	188	180
Heart Rate Range (bpm)	110	148
Resting Stroke Volume of left ventricle (cm^3)	75	183
Maximum Stroke Volume of left ventricle (cm^3)	80	166
Resting Cardiac Output (cm^3)	5850	5850
Maximum Cardiac Output (cm^3)	15040	29880
Heart Weight (g)	300	350
Total Heart Volume	800	1600

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

The study of the adaptation of the heart to exercise of different types in living subjects is limited by the means of measurement available. Initially it was done by simply listening to the heart and chest tapping, major advances included X rays, echocardiography *(using ultra sound)*, two dimensional echocardiography, and magnetic resonance imaging *(MRI)*. As techniques are refined the resolution and 3D capabilities are continually improved. It is important to know whether the images of the heart chambers are of systolic or diastolic end volumes, therefore they should be ECG triggered.

That the heart increases in size and efficiency as a result of prolonged physical training seems self evident. Greater demands on the heart would be expected to result in its enlargement to a greater or lesser extent *(cardiac hypertrophy)*.

Continued....

Earlier techniques could only assess left ventricle dimensions, so that in all major texts only left ventricular hypertrophy is referred to. This can give the impression that the enlargements are restricted to the left ventricle.

However, as the right side of the heart supplies the left side, the cardiac output of the right side must match that of the left side, othewise an imbalance of blood flow would result. The changes in the heart therefore are symmetrical, with the possible exception of a greater thickening of the left ventricular walls, which are initially thicker in all subjects *(including sedentary subjects)* as a result of the greater force required to pump blood around the body, as opposed to the lungs. Pathological heart conditions can result in much greater enlargement, and different chambers are affected differently.

The largest hearts in absolute terms are found in endurance athletes with high lean body weight in events where body weight is not a direct disadvantage to the activity, eg. swimming, cycling *(except when going uphill)* and rowing. A range of 1000 - 1100 cm^3 has been reported, with the largest recorded overall size of heart being 1700 cm^3 in a cyclist - more than twice the average volume of 700 cm^3.

Along with these increases in dimensions, there is a marked slowing of the resting heart rate *(bradycardia)* and disturbances of ECG and heart rhythms.

Most work has been done on males and there is little research data on females to date.

Endurance athletes show an increase in heart wall thickness and internal chamber diameter, whilst it is reported that resistance trainers show a mild thickening of the left ventricle wall and no significant increase in cavity size.

In measurements of 1000 athletes and 800 controls, average increases of left ventricle wall thickness of 1.6 mm, and increases of left ventricle end diastolic diameter of 5.3 mm have been reported. Where normal values are of 8 - 12 mm and 40 - 54 mm respectively.

Upper measures in athletes are a left ventricular thickness of 14 mm, and left ventricle end diastolic diameter of 70 mm.

Estimates of the time taken for these adaptations to develop vary from weeks, to months, to years. Similarly there are conflicting reports of the reversibility of the changes with the cessation of activity. Otherwise it appears that minimal training levels can maintain the adaptations.

However, there are some important criticisms of this accepted wisdom on athlete's heart.

When the heart measurements are standardised against lean body mass, increases are often relatively small and not universal. Absolute cardiac dimensions being more closely related to lean body mass than to type or state of training.

Continued....

Also some claim that the reported average enlargements in the trained heart are below the limits of resolution of echocardiography. It is also suggested that greater diastolic filling of the ventricles occurs rather than a true growth in ventricular size. This is such a controversial claim that the reference is provided below.

(Exercise Induced Cardiac Hypertrophy - Fact or Fallacy. Perrault H & Turcotte R A. Sports Medicine 17(5) 288 - 308. 1994.)

Twin studies suggest that hereditary factors are important in determining heart dimensions, and/or degree of cardiac adaptability to training, to an extent from 25 - 70% *(although it must be noted that others contradict these findings, reducing the estimate of the influence of heredity)*.

Interestingly, Greyhounds have relatively large hearts in relation to their lean body mass, and this does not vary significantly whether they are sedentary, racing, or retired. Thus suggesting that the relatively large hearts of greyhounds are the result of centuries of selective inbreeding, not of training enlargement.

These observations suggest that cardiac enlargement in athletes may be primarily determined by heredity. In other words, that endurance athletes are endurance athletes because of their relatively enlarged hearts, rather than endurance athletes have relatively large hearts as a result of their training; and if there is a training adaptation then the ability to adapt in this way is itself determined genetically.

Resting heart rate reflects the size of the stroke volume. As the metabolic demands of the two are approximately the same, the cardiac output at rest for the two is approximately the same, and therefore the smaller heart with its smaller stroke volume will have to beat faster to achieve the same cardiac output as the larger, more efficient heart.

Maximum heart rate decreases with age, and with long periods of endurance work, as a result of an increase in stroke volume and therefore longer time for diastolic filling, possibly due partly to the development of athlete's heart.

Heart rate range gives a measure of the amount by which the heart rate can increase over that at rest. Assuming a proportional increase in cardiac output *(at least up to close to the maximum heart rate)*, a greater heart rate range will mean that the athlete will have a greater capacity to adapt to greater aerobic demands.

Resting and Maximum stroke volumes of each ventricle involve both the size of the internal volume of the ventricle at the end of diastole *(i.e. when the ventricles are relaxed and completely filled)* and the ejection fraction *(i.e. the amount of the end diastolic blood volume that is actually ejected from the ventricle)*, which in turn is a function of the degree of contractility of the cardiac muscle fibres in the ventricle wall. As with all heart capacities it is common usage to refer to them in terms of the volume of blood ejected into the aorta from the left ventricle. However, the output of the right ventricle to the lungs matches that of the left ventricle to the body, otherwise the circulation could not be maintained.

Continued....

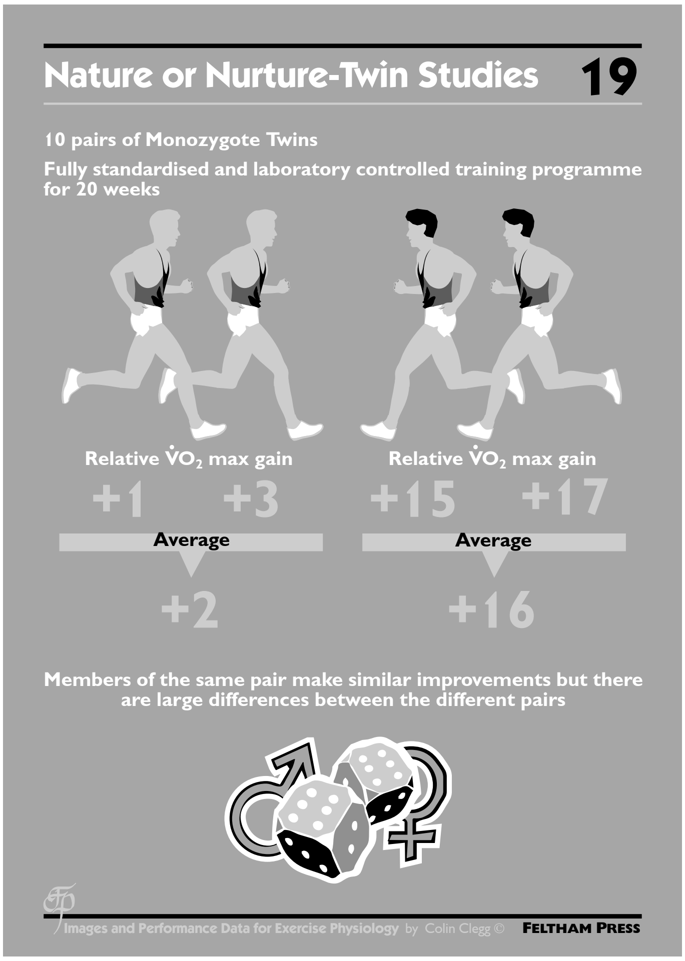
Resting and Maximum Cardiac Outputs are the products of the corresponding heart rates and stroke volumes, with the reservation that at very high heart rates the ventricles may not be filled completely during the shortened diastole.

Heart weight varies with body weight, generally increasing proportionally with the lean body weight, with an upper limit of about 500 g. This relationship with lean body weight complicates any analysis of the increase in heart weight as a direct result of endurance training.

Total heart volume also varies with body weight, with a maximum of 1700 cm^3 being quoted for a professional cyclist. Overall the influence of body size is an important factor, that must always be taken into account.

Nature or Nurture - Twin Studies

Image 19



Nature or Nurture-Twin Studies 19

10 pairs of Monozygote Twins

Fully standardised and laboratory controlled training programme for 20 weeks

Relative $\dot{V}O_2$ max gain

+1 +3

Average

+2

Relative $\dot{V}O_2$ max gain

+15 +17

Average

+16

Members of the same pair make similar improvements but there are large differences between the different pairs

Images and Performance Data for Exercise Physiology by Colin Clegg © FELTHAM PRESS

Twin studies always provide fascinating insights into the ‘nature v nurture’ debate on the relative contribution of inherited capacities and training regimes to successful sporting performances.

Identical twins develop from a single fertilised egg, which gives rise to two genetically identical individuals. Although it must be remembered that environmental differences will immediately exert their influence within the uterus of the mother, so that even by birth all other things may not be equal.

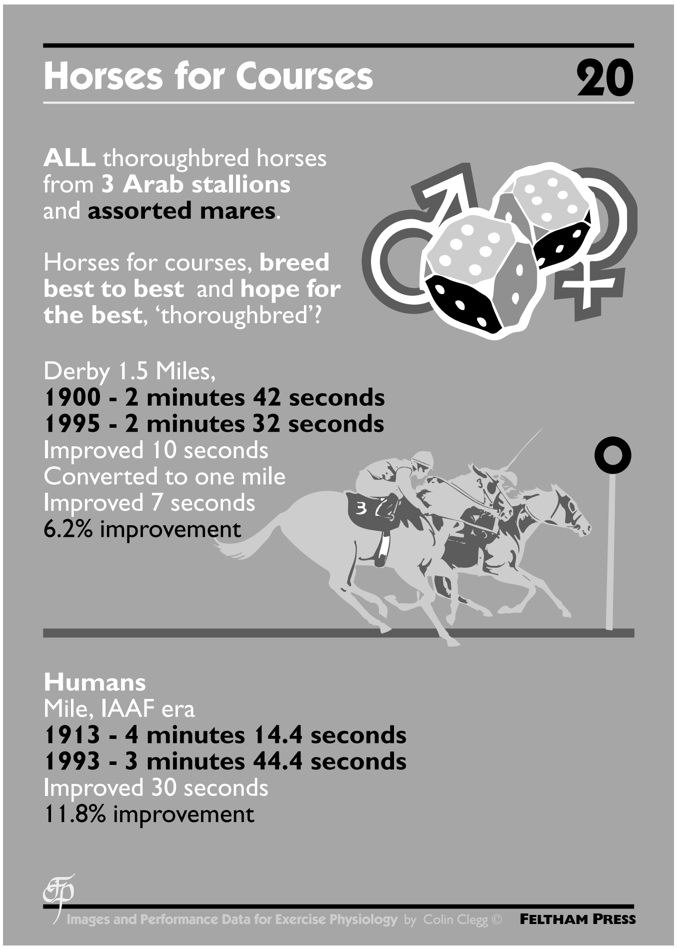
Before the advent of DNA fingerprinting it was also very difficult to be sure that twins were identical, as opposed to being non-identical *(arising from a double fertilisation of two eggs)*. This uncertainty could undermine earlier twin studies.

Differences in ‘nurture’ or environment will lead to variation between members of pairs of identical twins, but as the figures show, these differences will be less than those between members of different pairs of identical twins. The figures shown indicate that the ability to improve through training is itself influenced strongly by inheritance.

To consider the predetermined genetic component of sporting ability, environmental influences must be optimal, as obviously inherent capacities can only be developed in optimum environmental conditions.

Horses for courses

Image 20



Horses for Courses **20**

ALL thoroughbred horses
from **3 Arab stallions**
and **assorted mares**.

Horses for courses, **breed**
best to best and **hope for**
the best, 'thoroughbred'?

Derby 1.5 Miles,
1900 - 2 minutes 42 seconds
1995 - 2 minutes 32 seconds
Improved 10 seconds
Converted to one mile
Improved 7 seconds
6.2% improvement

Humans
Mile, IAAF era
1913 - 4 minutes 14.4 seconds
1993 - 3 minutes 44.4 seconds
Improved 30 seconds
11.8% improvement

Images and Performance Data for Exercise Physiology by Colin Clegg © **FELTHAM PRESS**

A consideration of race horses introduces an unusual and interesting new perspective to the study of training and fitness.

Race horse selection is firmly based on the belief that breeding, i.e. the inheritance of the necessary physical and physiological attributes for speed and endurance, is the chief pre-requisite for success, without which all the best training in the world is ineffective.

However, as the figures show, despite all this selective breeding, there has been relatively little improvement over the years in the performance of race horses.

With humans, the proportion of inherited variation which contributes to sporting success is typically downplayed, despite glaring examples to the contrary. There is typically much talk by coaches and athletes of training 'secrets', the 'will to succeed', nutrient supplementation, and a myriad other unquantifiable components. Even a Kenyan world record holder has allegedly been quoted as saying: "we are not born champions - it is the training that we do".

Continued....

Further claims that the 'Africans' predominate at so many running distances because they "used to run to school" reveal much about the state of education in this still 'mysterious' subject of exercise physiology, and should lead to lively debate. As should an alleged quote of a recently appointed National Coach for British runners that: "I tell my runners that they have got two arms and two legs just like all the others, and to go out there and compete". One wonders about the importance of the bits inbetween.

Consideration of the figures presented should lead to much debate on the dilemma of nature and/or nurture.

Certainly the relative lack of improvement in race horses could be taken to indicate that within a relatively small 'gene pool' of a highly selected group, the room for improvement through training innovations is limited. An observation supported by human twin studies.

The relatively large improvement by humans can be seen as a result of the ever increasing participation rates. Certainly up to the 1950's, athletics and many other sports were largely the preserve of a privileged elite.

There are interesting differences in the training regimes of horses and humans which could repay investigation. For example horses are rarely trained over distances longer than that to be raced, whereas long slow distance running has and does play a large part in training regimes of humans, even that of 800 m runners. Arthur Lydiard, coach of 800 m Olympic Gold Medalist and World Record Holder Peter Snell, and others, based his training on long slow distance running. Although increasingly the emphasis is now on speed and intensity with adequate rest. Horses on a training regime of alternating hard and rest days, could sustain increased work loads on the hard days, but showed symptoms of overtraining if the work load was increased on the 'rest' days.

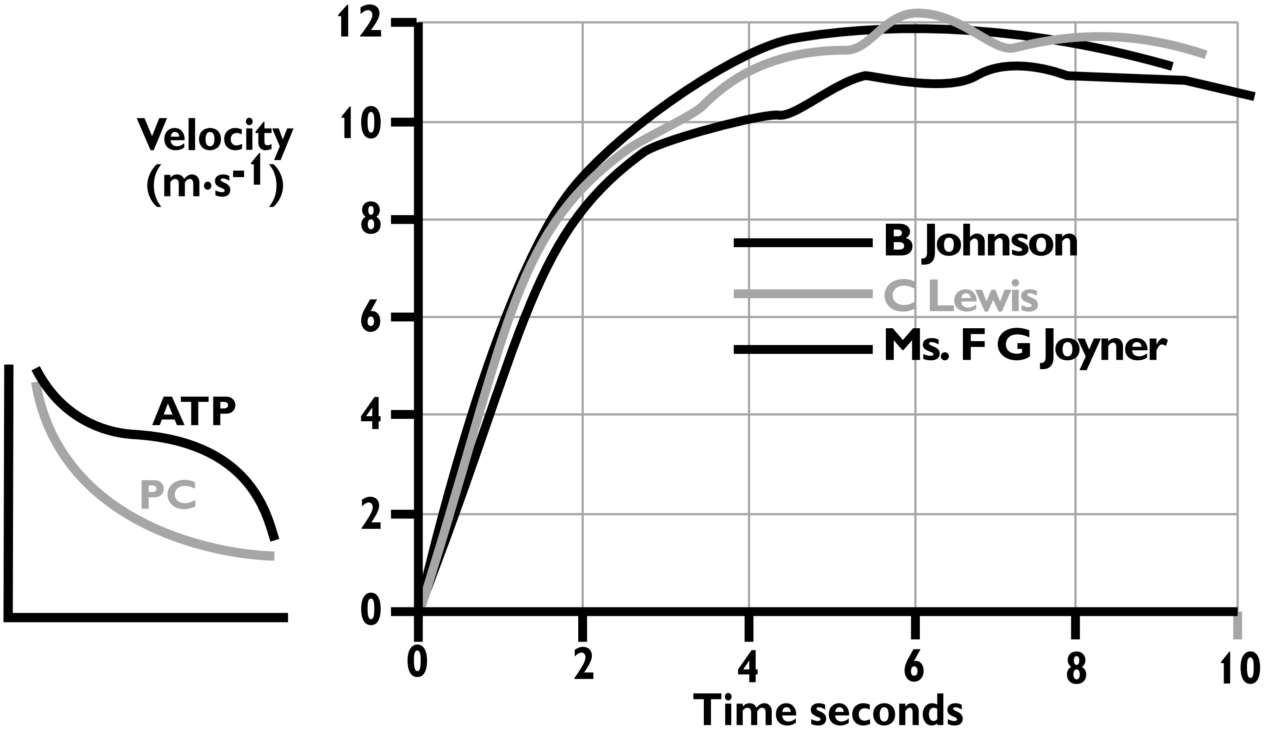
Nor do horses carry out extensive stretching regimes so beloved of human runners!

APPENDIX 1

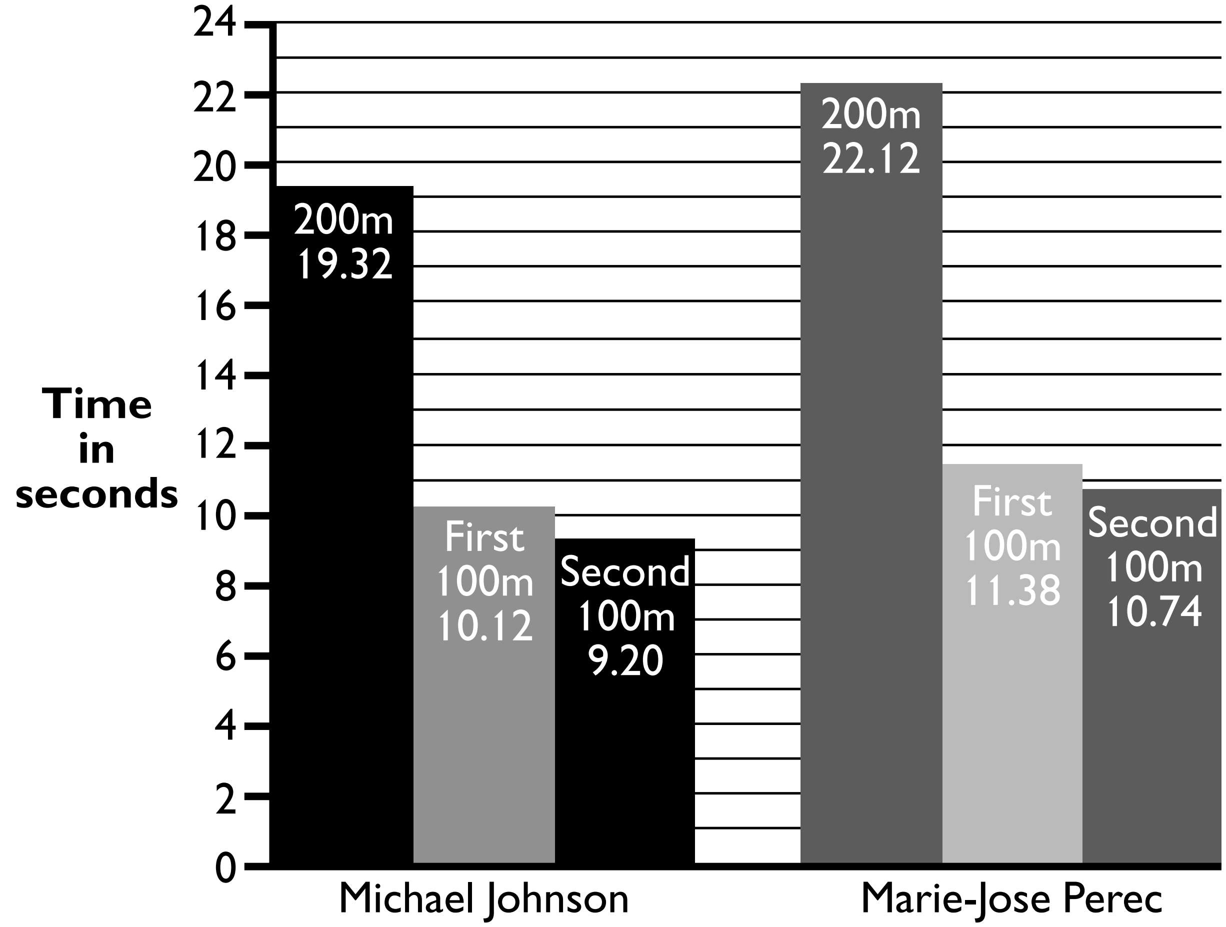
100 metres Olympic Finals 1

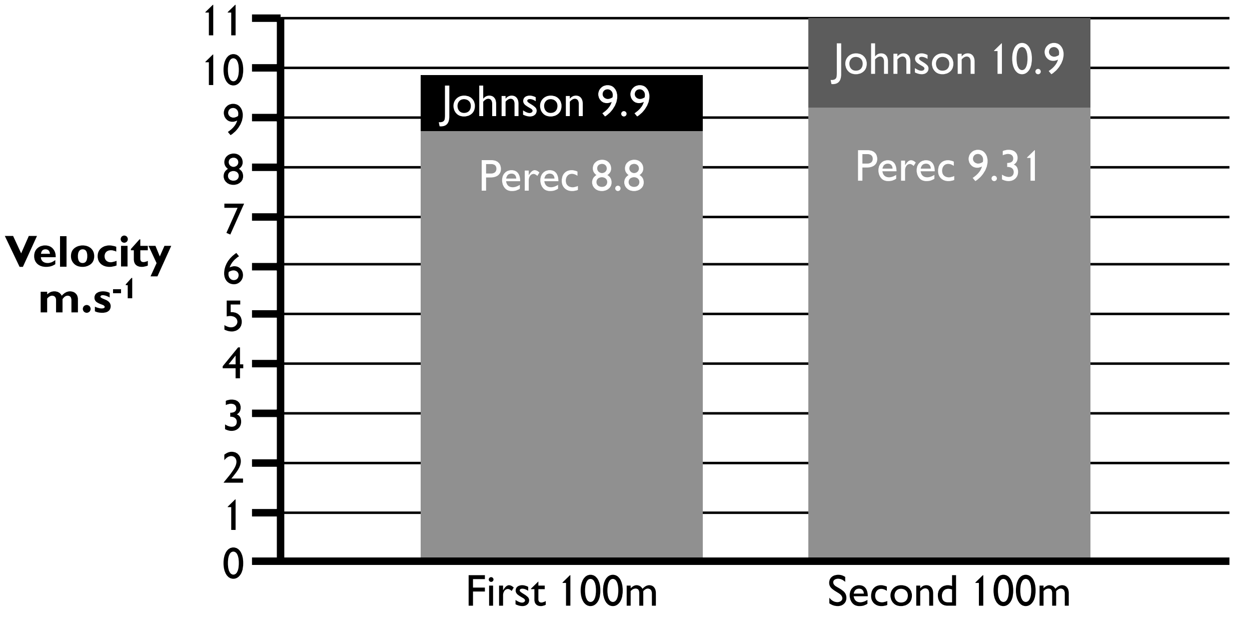
Data from IAAF Biomechanical Analysis Programme 1988 Seoul Olympics 100m Finals

Distance metres	B Johnson seconds	C Lewis seconds	Ms. F G Joyner seconds
10	1.83	1.89	2.00
20	2.87	2.96	3.09
30	3.80	3.90	4.09
40	4.66	4.79	5.06
50	5.50	5.65	5.97
60	6.33	6.48	6.89
70	7.17	7.33	7.80
80	8.02	8.18	8.71
90	8.89	9.04	9.62
100	9.79	9.92	10.54



200 metres


Time in seconds
24
22
20
18
16
14
12
10
8
6
4
2
0
200m 19.32
First 100m 10.12
Second 100m 9.20
200m 22.12
First 100m 11.38
Second 100m 10.74
Michael Johnson
Marie-Jose Perec


Velocity m.s-1
11
10
9
8
7
6
5
4
3
2
1
0
Johnson 9.9
Perec 8.8
Johnson 10.9
Perec 9.31
First 100m
Second 100m

400 metres World Records *(Men)* 3

Accumulative 100 m splits

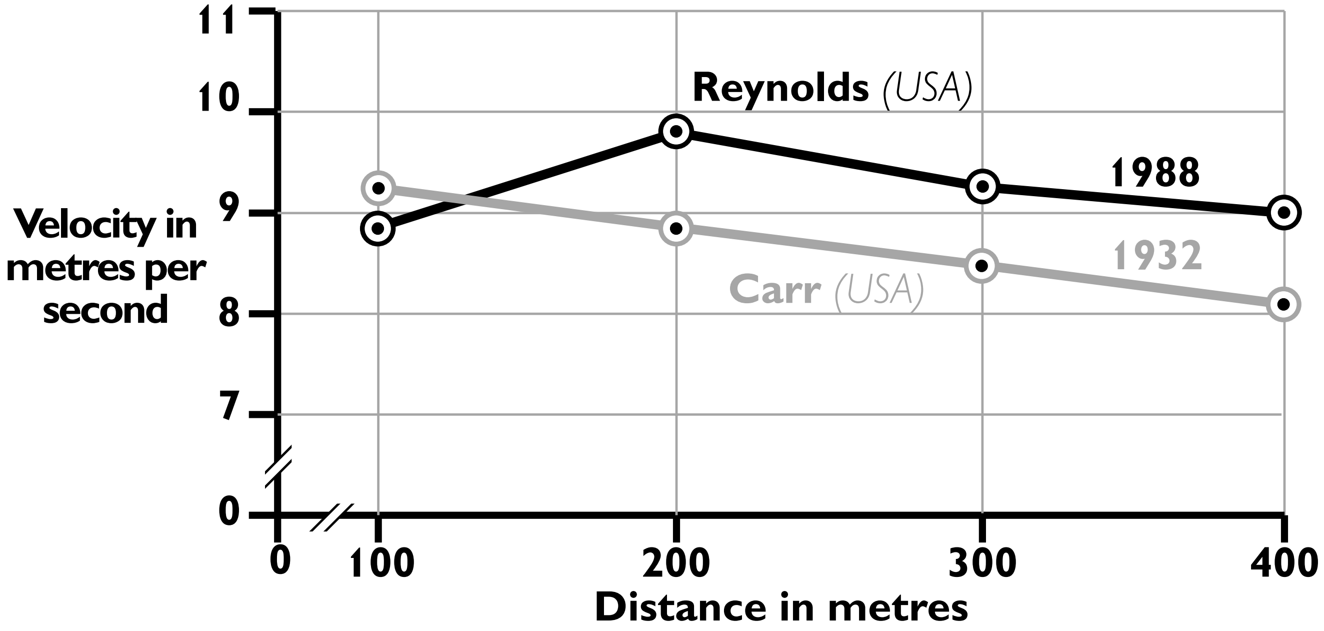
Name	Date	100 m	200 m	300 m	400 m
Carr *(USA)*	1932	10.90	22.10	33.80	46.20
Harbig *(Germany)*	1939	11.30	22.00	33.60	46.00
Klemmer *(USA)*	1941	10.80	21.80	33.40	46.00
Smith *(USA)*	1967	11.00	21.70	33.50	44.50
Evans *(USA)*	1968	10.40	21.10	32.20	43.86
Reynolds *(USA)*	1988	11.20	21.40	32.20	43.29

Times for each 100 m

Name	Date	100 m	200 m	300 m	400 m	Even Pace
Carr *(USA)*	1932	10.90	11.20	11.70	12.40	11.55
Harbig *(Germany)*	1939	11.30	10.70	11.60	12.40	11.50
Klemmer *(USA)*	1941	10.80	11.00	11.60	12.60	11.50
Smith *(USA)*	1967	11.00	10.70	11.80	11.00	11.13
Evans *(USA)*	1968	10.40	10.70	11.10	11.66	10.97
Reynolds *(USA)*	1988	11.20	10.20	10.80	11.09	10.82

Velocity in metres per second *(rounded up)* for each 100 m

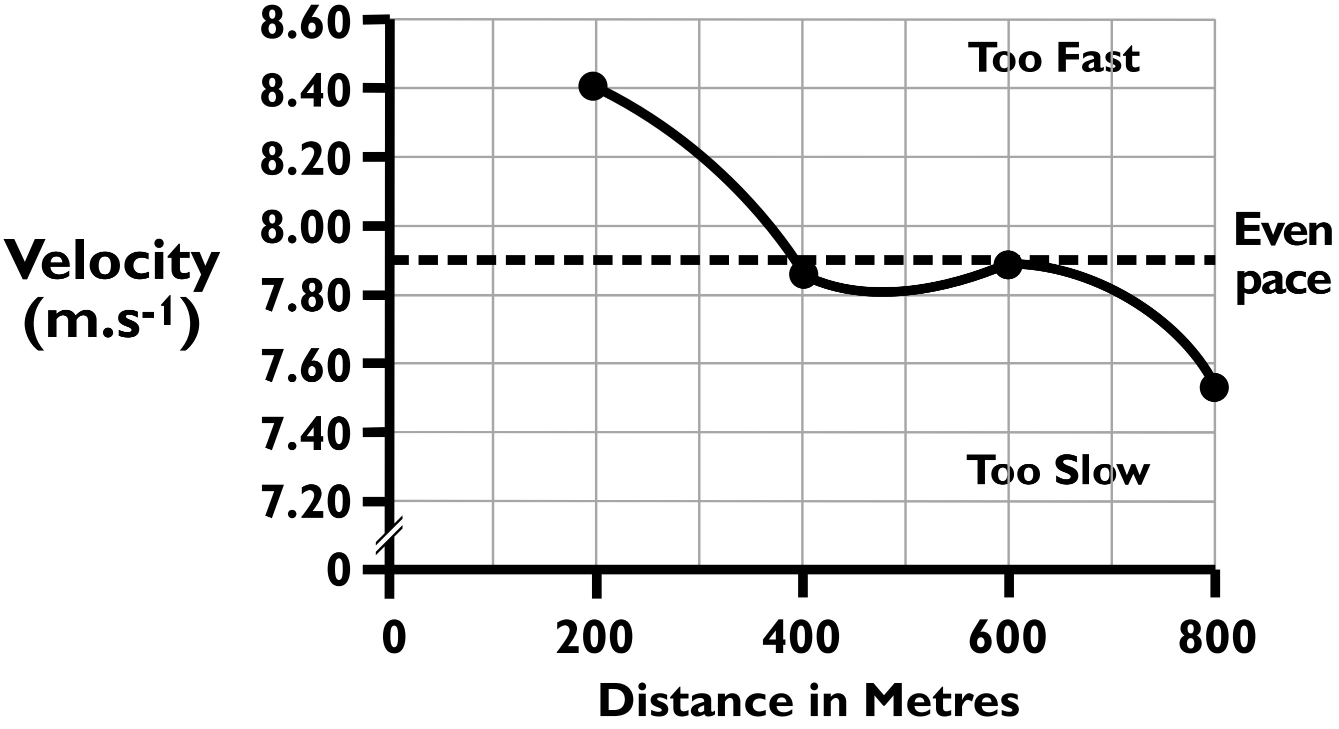
Name	Date	100 m	200 m	300 m	400 m	Even Pace
Carr *(USA)*	1932	9.2	8.9	8.5	8.1	8.7
Harbig *(Germany)*	1939	8.9	9.3	8.6	8.1	8.7
Klemmer *(USA)*	1941	9.3	9.1	8.6	7.9	8.7
Smith *(USA)*	1967	9.1	9.3	8.5	9.1	9.0
Evans *(USA)*	1968	9.6	9.3	9.0	8.6	9.1
Reynolds *(USA)*	1988	8.9	9.8	9.3	9.0	9.2



800 metres World Record *(Men)* 4

Men's 800m World Record Kipketer 1.41.11

Distance	200m Splits Seconds	Velocity ($m.s^{-1}$)
Even pace	25.28	7.91
200m	23.8	8.40
400m	25.5	7.84
600m	25.3	7.90
800m	26.5	7.55



Seb Coe's 800m World Record of 1m 41.73 in 1981 lasted until....

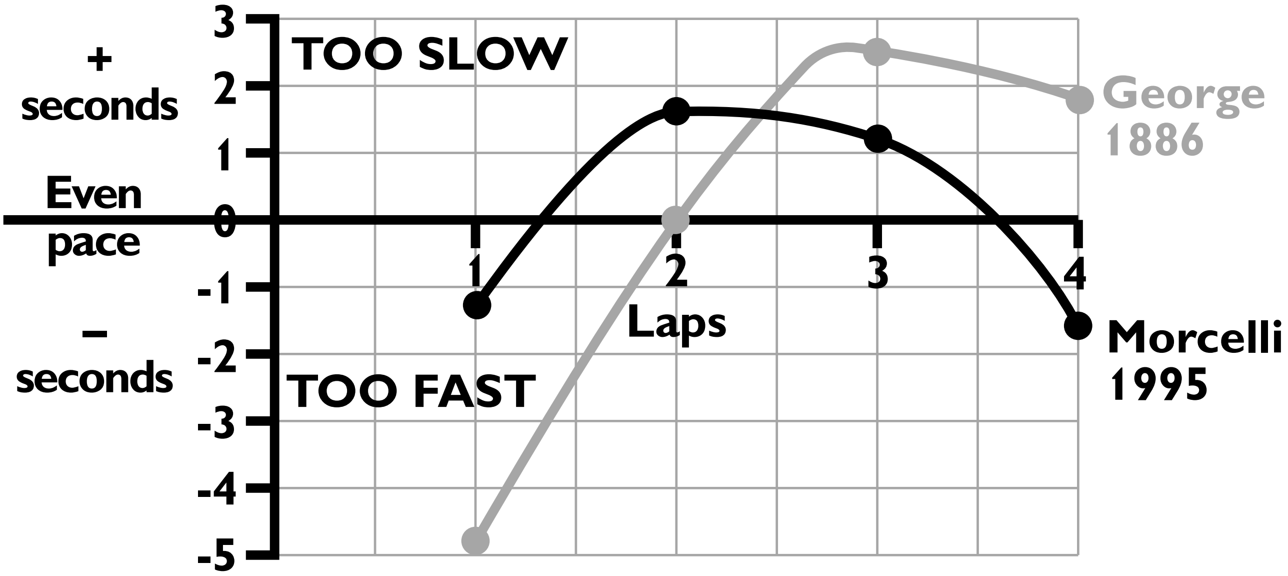
1997 when broken by Kipketer *(Kenyan)* 1m 41.11

16 years to improve by 0.62s.

Mile World Records

Men's Mile Record & Lap *(440 yards)* Times at approximately10 year intervals

	Time	Lap 1	Lap 2	Lap 3	Lap 4	Even pace
1886 George	4.12.75	58.50	63.20	65.75	65.00	63.18
1915 Taber	4.12.60	58.00	67.00	68.00	59.60	63.15
1923 Nurmi	4.10.40	60.30	62.90	63.50	63.70	62.60
1933 Lovelock	4.07.60	61.40	62.20	65.10	58.90	61.90
1943 Anderson	4.02.60	58.50	61.50	63.50	60.60	60.65
1954 Bannister	3.59.40	57.50	60.70	62.30	58.90	59.85
1964 Snell	3.54.10	56.40	57.70	60.20	59.80	58.53
1975 Bayi	3.51.00	56.90	59.70	58.70	55.70	57.75
1985 Cram	3.46.32	57.50	57.40	58.40	53.00	56.58
1995 Morcelli	3.44.39	54.90	57.70	57.30	54.50	56.09



1953 Bannister ran first sub 4 minute mile.

1997 Kohmen *(Kenyan)* ran two sub 4 minute miles one after the other in a two mile race.

Sustained the output for twice the distance.

1886 George ran mile in 4m 12.8s
Current men's world record is now 3m 44.39s.

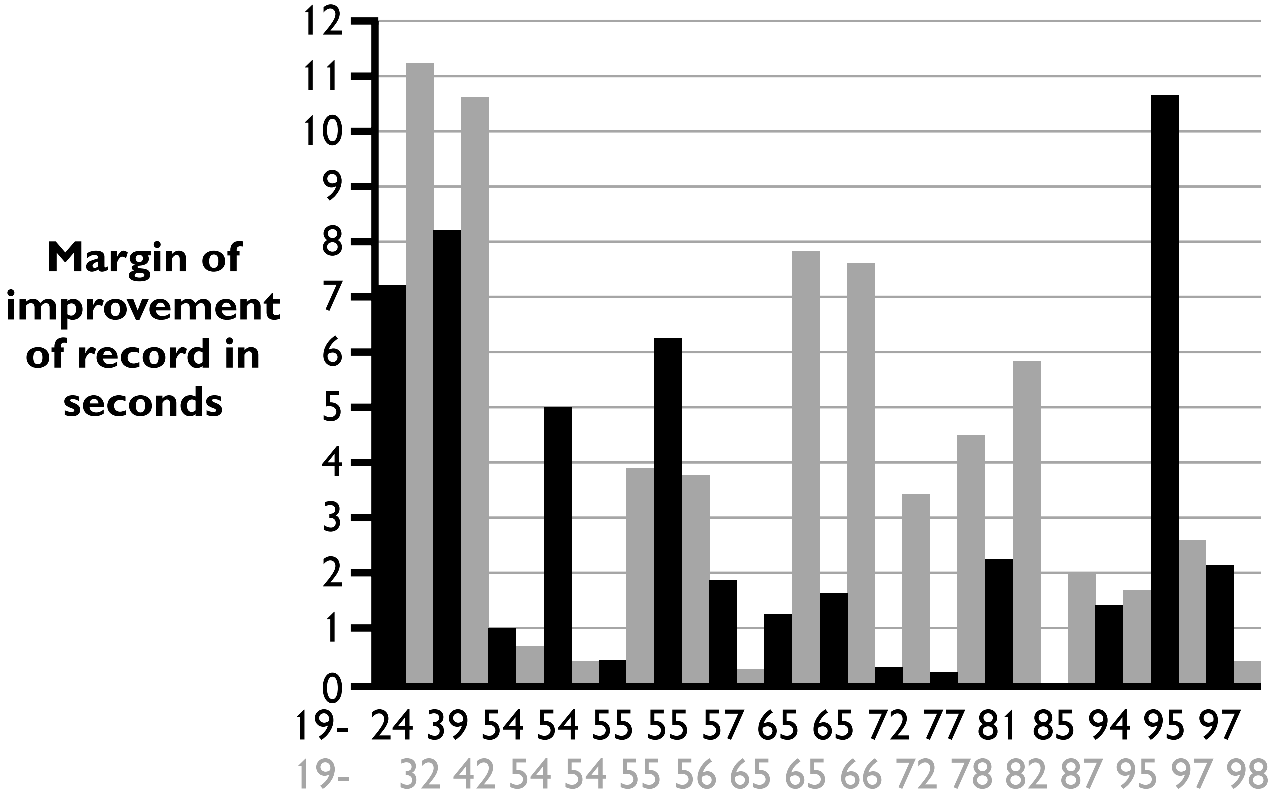
George's time of 1886 not beaten by a woman until:

Masterkova in 1996 4m 12.56s

5000 metres World Records *(Men)* 6

1 kilometre splits in selected World Records

Time	Name	Year	1k	2k	3k	4k
14:36.60	**Kolehmained** *(Finland)*	**1912**	**-**	**5:47.00**	**8:46.00**	**-**
14:35.40	**Nurmi** *(Fin)*	1922	2:43.00	5:36.10	8:35.80	11:37.70
14:17.00	**Lehtinen** *(Fin)*	**1932**	**2:46.50**	**5:40.50**	**8:36.00**	**11:33.50**
14:08.80	**Maki** *(Fin)*	1939	2:46.00	5:39.00	8:32.50	11:24.50
13:58.20	**Hagg** *(Sweden)*	**1942**	**2:40.00**	**5:27.00**	**8:18.50**	**11:09.00**
13:57.20	**Zatopek** *(Czech)*	1954	2:47.10	5:34.10	8:23.50	11:13.40
13:46.80	**Kuts** *(USSR)*	**1955**	**2:42.00**	**5:30.00**	**8:16.00**	**10:06.00**
13:36.80	**Pirie** *(GB)*	1956	2:36.00	5:22.00	8:09.00	10:57.00
13:25.80	**Clarke** *(Australia)*	**1965**	**2:39.10**	**5:20.40**	**8:04.10**	**10:48.70**
13:16.60	**Clarke** *(Australia)*	1966	2:40.20	5:16.40	7:57.40	10:39.00
13:00.41	**Moorcroft** *(GB)*	**1982**	**2:38.00**	**5:12.60**	**7:50.20**	**10:28.70**
13:00.40	**Aouita** *(Morocco)*	1985	2:35.14	5:13.82	7:51.00	10:32.16
12:58.39	**Aouita** *(Morocco)*	**1987**	**2:35.35**	**5:13.03**	**7:46.37**	**10:26.05**
12:44.39	**Gebrselassie** *(Ethiopia)*	1995	2:34.04	5:00.70	7:42.92	10:14.15
12:39.74	**Gebreselassie** *(Ethiopia)*	**1998**	**2:43.80**	**5:06.40**	**7:43.80**	**10:12.10**



10 000 metres *(Men)*

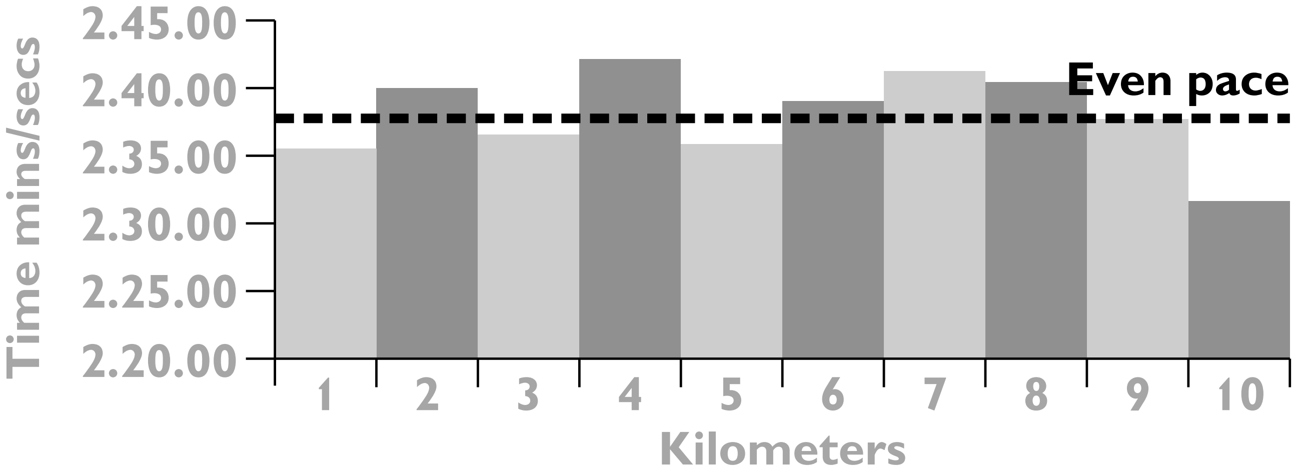
Gebreselassie 10 000m - time 26.22.75

	'Splits'	Km times	Km pace
First kilometer		2.35.36	
		5.15.53	2.40.17
		7.53.03	2.37.50
		10.35.03	2.42.00
First 5 000m	13.11.53	13.11.53	2.36.50
		15.50.97	2.39.44
		18.32.45	2.41.48
		21.12.92	2.40.47
		23.51.53	2.38.61
Second 5 000m	13.11.22	26.22.75	2.31.22

Even pace
Km 2.38.28
Laps 63.31s

Equivalent
mile pace
4.15.28

Velocity of
6.32 m·s^{-1}



5 000m splits 13.11.53 & 13.11.22 both better than Olympic 5 000m record up until 1978.

Both 5 000m splits of Gebreselassie's 10 000m world record would have been a 5 000m world record up until 1978 when Rono ran 13.08.04

Both 5 000m splits would have also won all Olympic 5 000m finals except for:
1984 Aouita 13.05.59 O.R., & 1996 Niyongabo 13.07.96.

Although of course Olympic finals are tactical.

Image removed as crucial information is lost in black and white.

Differences between Men's and Women's Performances

Parity of women in ultra distance events.

Cycling

In 1967 Beryl Burton riding in the same event as the men overtook and beat Mick McNamara who was setting a new national record in the men's 12 hour time trial

1967 Best All Rounder (BAR) Competition

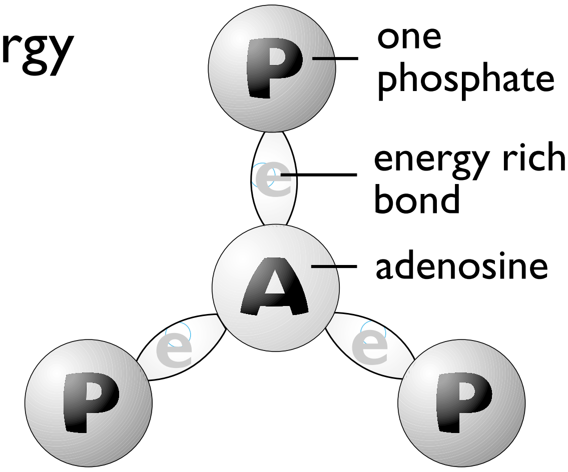
	50 Miles	100 Miles	12 Hours
Men's Mick McNamara	**1.52.25** **26.66 mph**	**3.54.45** **25.56 mph**	**276.52 Miles** **23.04 mph**
Women's Beryl Burton	**1.56.00** **25.86 mph**	**4.04.50** **24.49 mph**	**277.25 Miles** **23.10 mph**

In the same year the fastest men's 25 mile time trial was 52 mins 55 secs *(not part of men's BAR)* = 28.35 mph while Beryl Burton's was 56 mins 7 secs = 26.73 mph

Energy from ATP

10

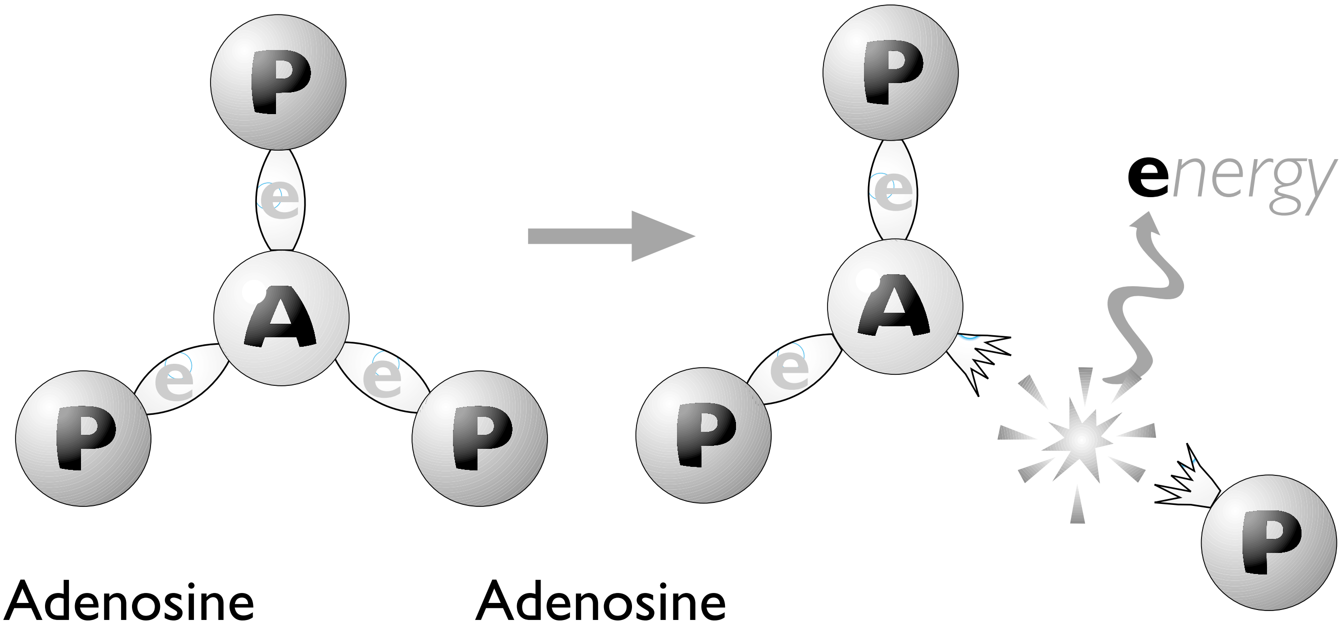
The body can only use energy in the form of **ATP**.


P
one phosphate
e
energy rich bond
A
adenosine
e
e
P
P

Adenosine **T**ri **P**hosphate has 3 phosphate groups.

If a phosphate group is removed from **ATP**, **energy** is released and **ADP** is formed.

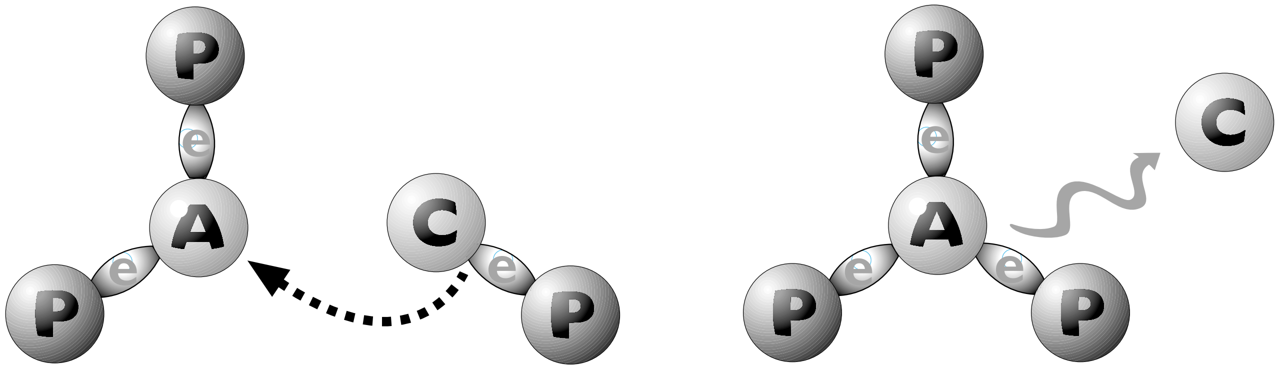
ADP has **2 phosphate** groups.


P
e
A
e
e
P
P
P
e
A
e
P
energy
P

Adenosine **Tri** Phosphate *(ATP)* → Adenosine **Di** Phosphate *(ADP)* + Phosphate *(P)* + Energy

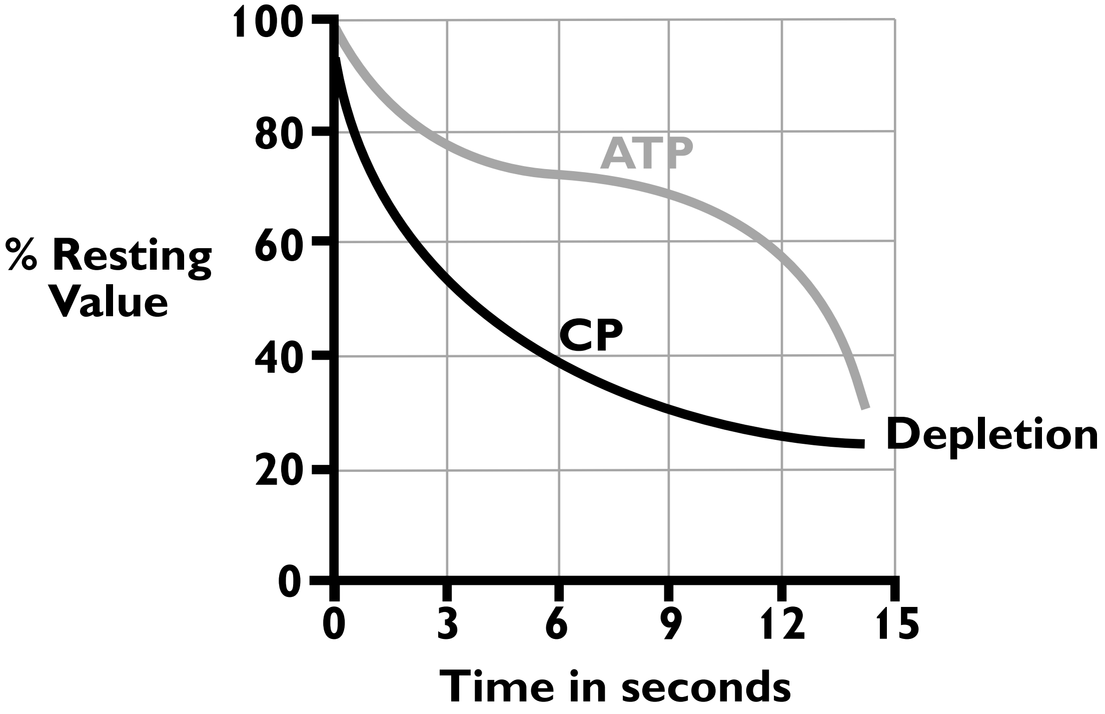
ATP Regeneration from Creatine Phosphate (CP)

The body only contains enough ATP for a few seconds of flat out activity
Then the ATP must be regenerated from the ADP using an energy reserve substance Creatine Phosphate (CP)



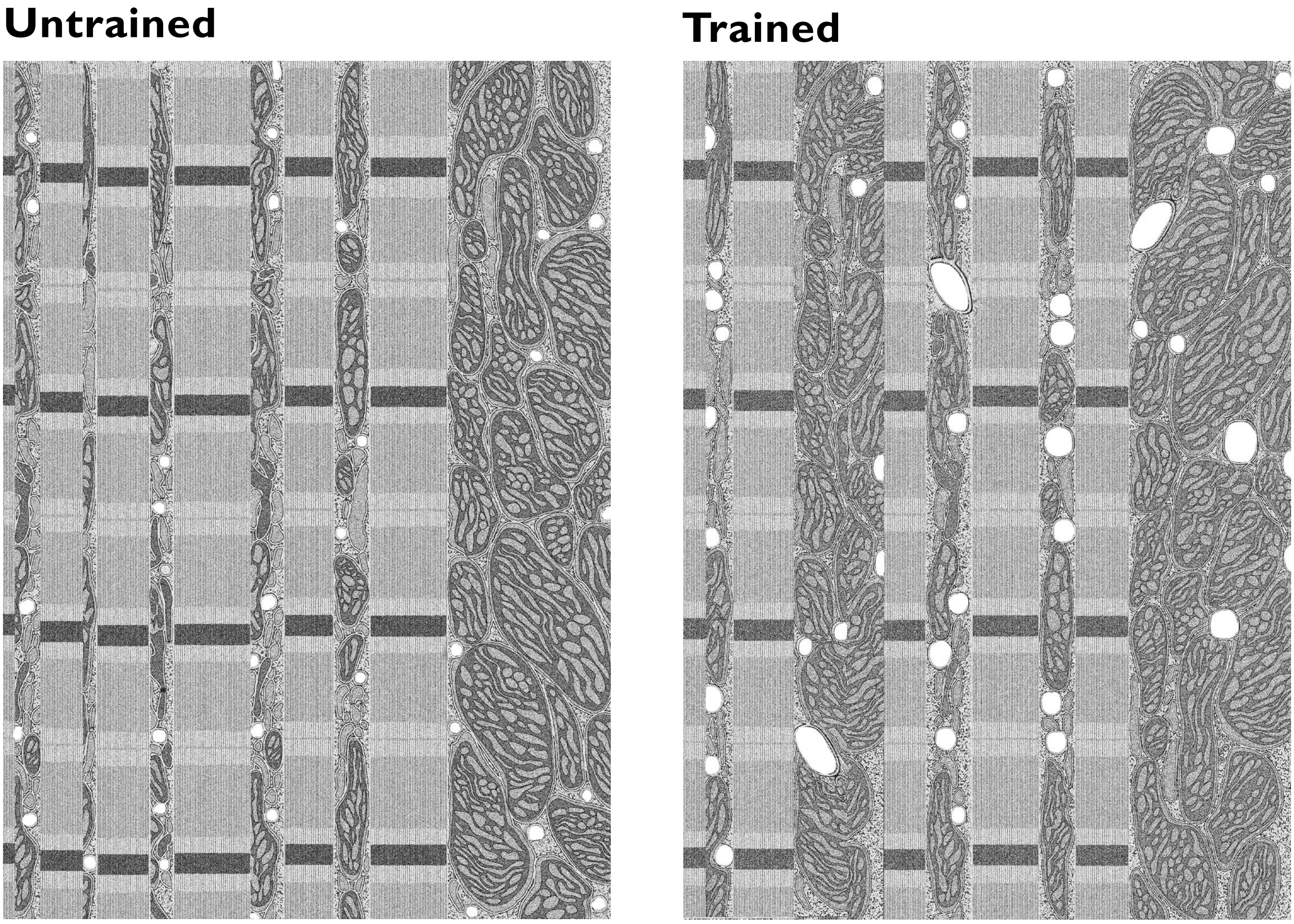
Adenosine **Di** Phosphate *(ADP)* + **Creatine Phosphate** ***(CP)*** → Adenosine **Tri** Phosphate *(ATP)* + **Creatine**

Levels of ATP and PC during a flat out sprint



Skeletal Muscle Myofibrils as Seen Under the Electron Microscope

12

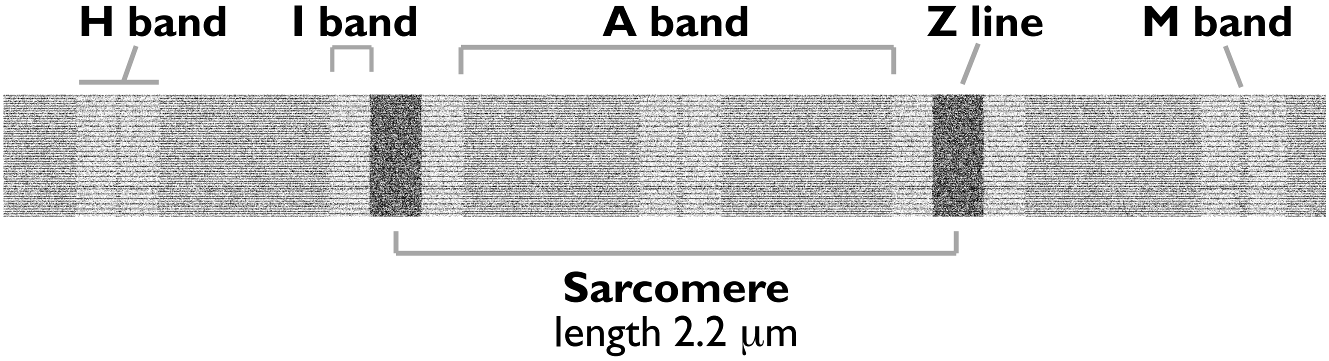


Untrained

Trained

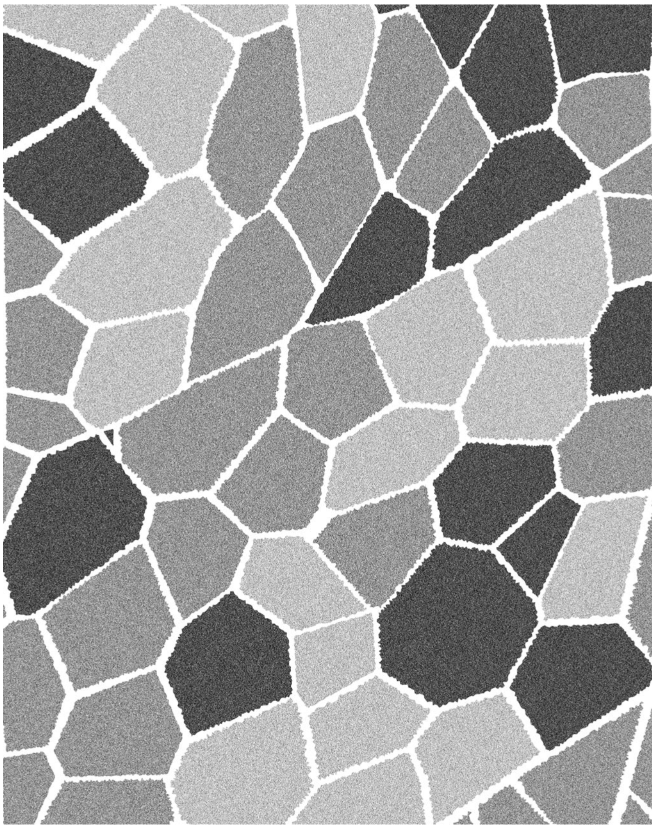
Note the increased size and number of mitochondria and fat vacuoles in the endurance trained muscle.

Magnification x5000

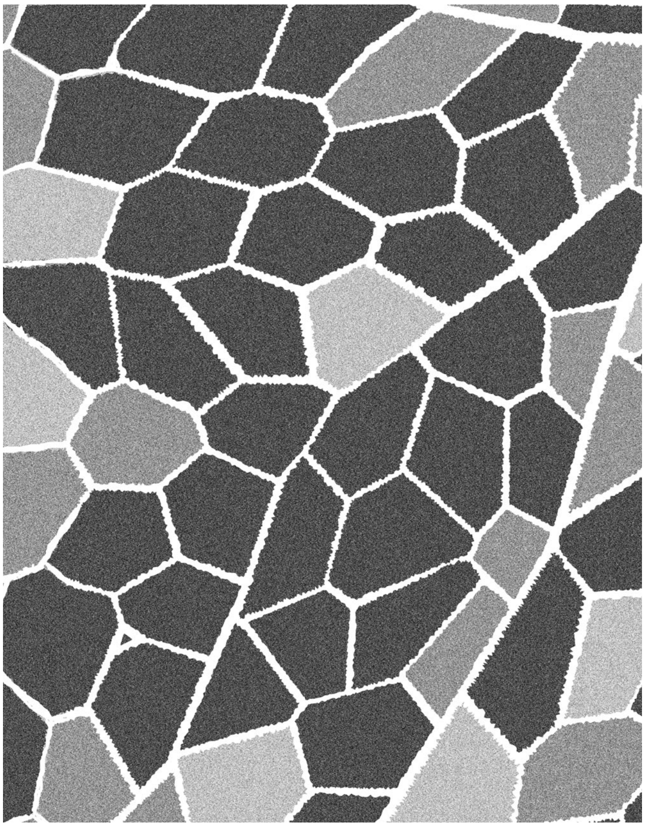

H band
I band
A band
Z line
M band
Sarcomere
length 2.2 μm

Slow and Fast Twitch Muscle Fibres in Humans

13



Sprint Runner
70% Fast-twitch fibres *(light)*



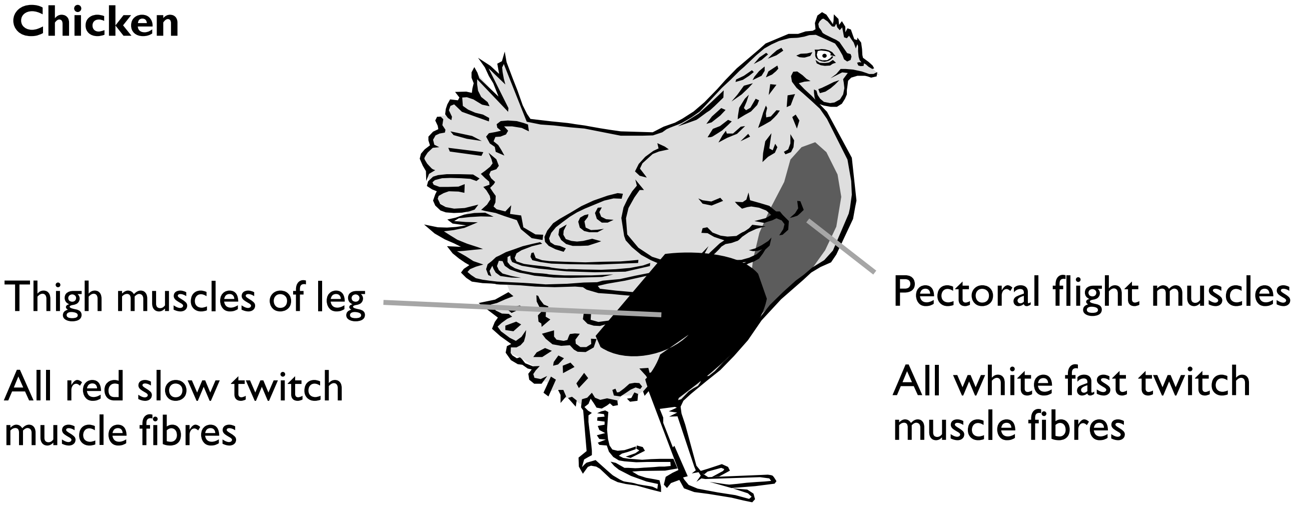
Marathon Runner
90% Slow twitch fibres *(dark)*

Skeletal Muscle Fibre Types and their Properties

Type	Colour	Contraction	Metabolism	Properties
I	Red	Slow-twitch	Oxidative (aerobic)	Fatigue resistant Low force/power
IIA	White	Fast-twitch	Oxidative/glycolytic (aerobic/anaerobic)	Intermediate
IIB	White	Fast-twitch	Glycolytic (anaerobic)	Fatigue susceptible High force/power

Slow and Fast Twitch Muscle Fibres in Other Animals

Chicken


Thigh muscles of leg
All red slow twitch muscle fibres
Pectoral flight muscles
All white fast twitch muscle fibres

Thigh muscles of leg

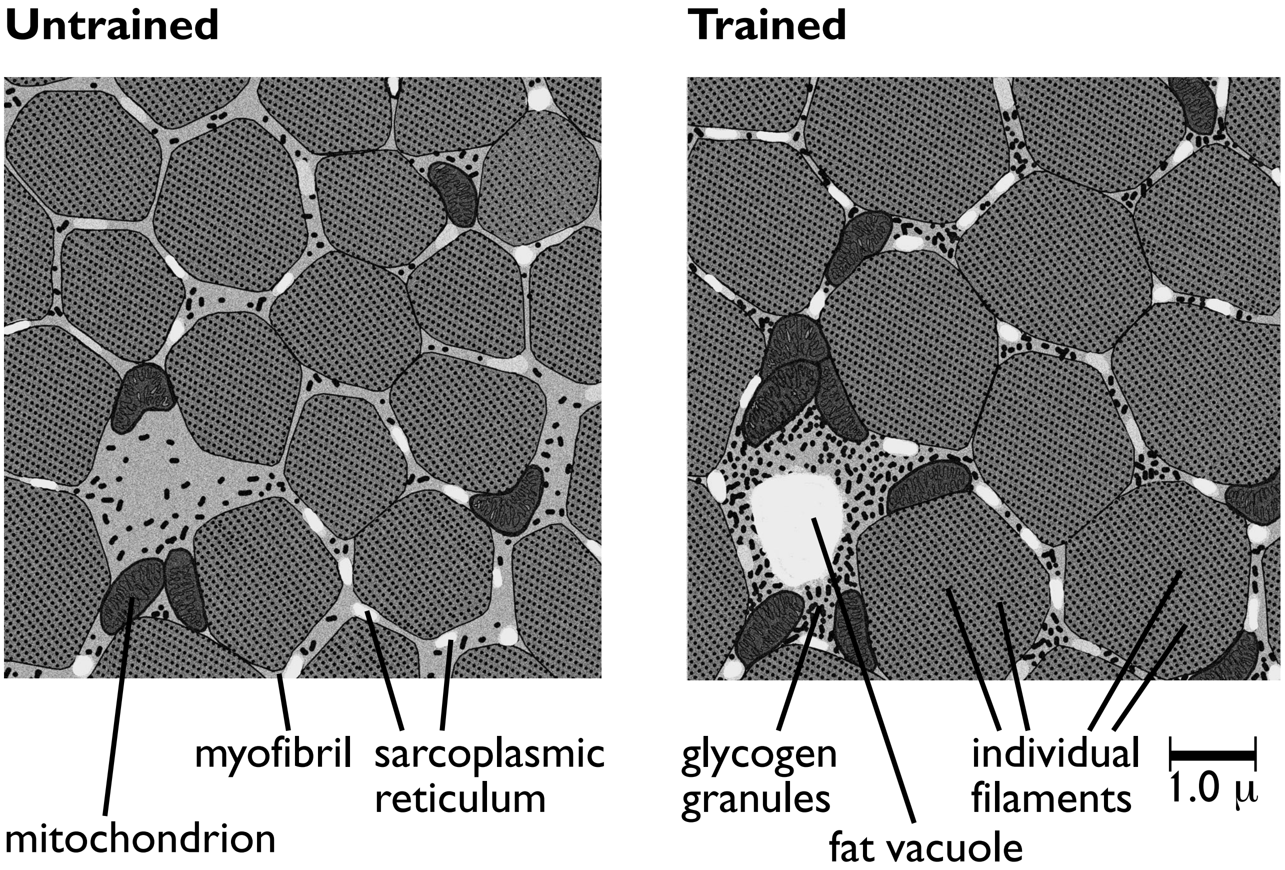
Pectoral flight muscles

Blood supply in fresh meat adds a reddish tinge to both.
The bright red colour of meat in the window of the butcher's shop
is due to an artificial additive to make it more 'attractive'.

Effect of Endurance Training on Slow Twitch Muscle Fibres

15

Effect of aerobic training on type I slow twitch muscle fibres. Mitochondria, myoglobin, glycogen stores, fat vacuoles, all increase. The figure shows these changes amongst the myofibrils within a single muscle fibre.


Untrained
Trained
myofibril
sarcoplasmic reticulum
mitochondrion
glycogen granules
individual filaments
fat vacuole
1.0 μ

Myoglobin - Average untrained 2g/100g muscle.

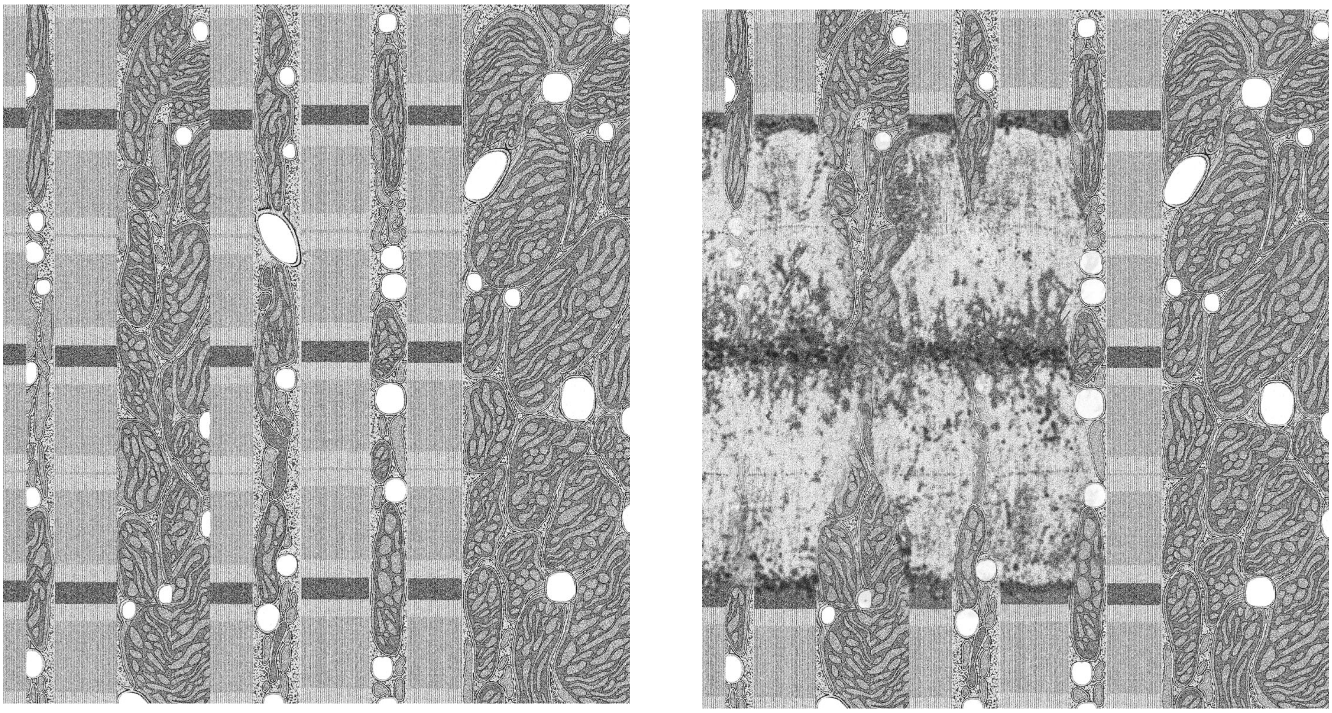
Myoglobin - Endurance trained 7g/100g muscle.

= 250% increase

16

Muscle Damage and Delayed Onset Muscle Soreness

Muscle damage after strenuous endurance work



Rested muscle

Muscle damaged by hours of running

Magnification x 5000

Note breakdown of integrity of fibres as a result of membrane damage.

Much of the damage caused by running occurs during the eccentric phases eg. quadriceps whilst running downhill.

Such damage results in an inflamatory reaction by the immune system.

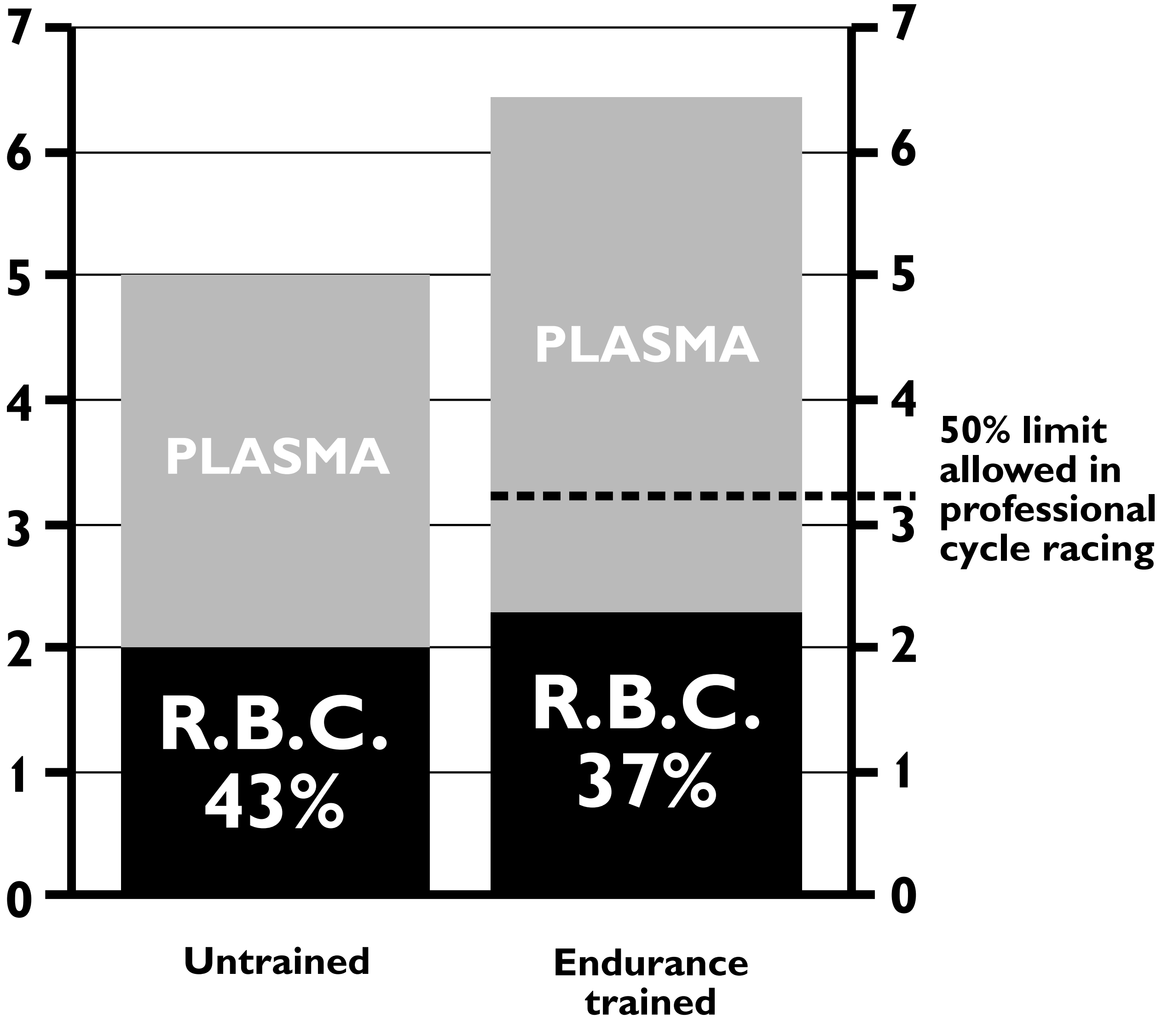
Effects of Endurance Training and Doping on Blood Composition

17

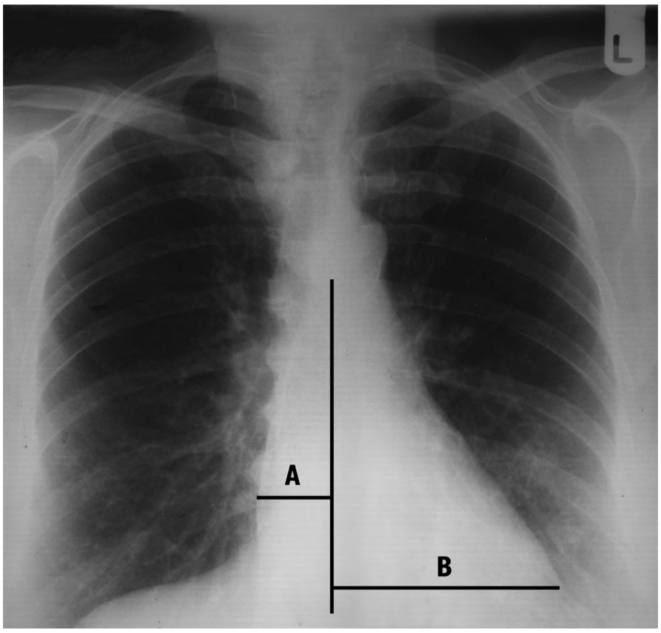
Endurance Athletes can experience Pseudo-anaemia

Due to increased plasma volume

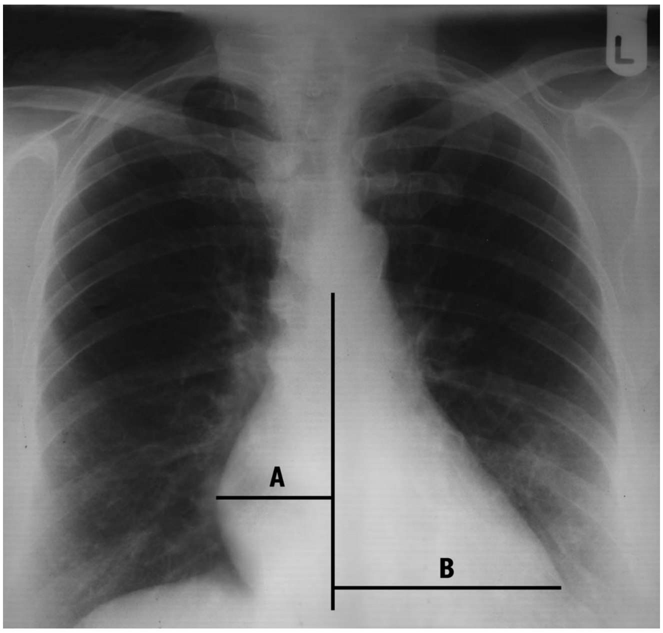
Conversely high red blood cell counts also occur



Heart Trained and Untrained 18



Average Male Heart Size



Endurance Athlete Heart Size

The cardiothoracic ratio is the sum of lines A and B divided by the largest transverse diameter of the chest (normally 0.50)

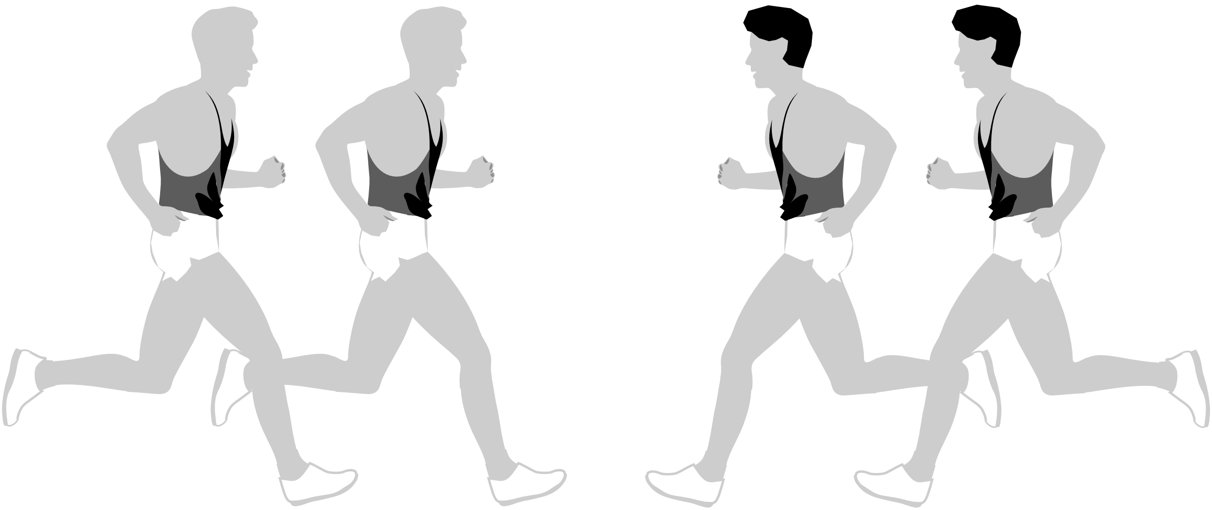
Cardiac Output (cm^3.min^{-1}) = Heart Rate (bpm) x Stroke Volume (cm^3)

	Normally Active Male	**Elite Male Marathon Runner**
Resting Heart Rate (bpm)	78	32
Maximum Heart Rate (bpm)	188	180
Heart Rate Range (bpm)	110	148
Resting Stroke Volume of left ventricle (cm^3)	75	183
Maximum Stroke Volume of left ventricle (cm^3)	80	166
Resting Cardiac Output (cm^3)	5850	5850
Maximum Cardiac Output (cm^3)	15040	29880
Heart Weight (g)	300	350
Total Heart Volume	800	1600

Nature or Nurture-Twin Studies 19

10 pairs of Monozygote Twins

Fully standardised and laboratory controlled training programme for 20 weeks



Relative $\dot{V}O_2$ max gain

+1 +3

Average

+2

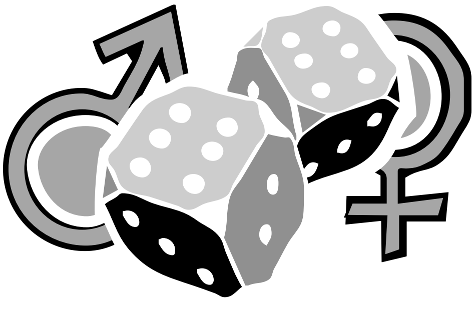
Relative $\dot{V}O_2$ max gain

+15 +17

Average

+16

Members of the same pair make similar improvements but there are large differences between the different pairs



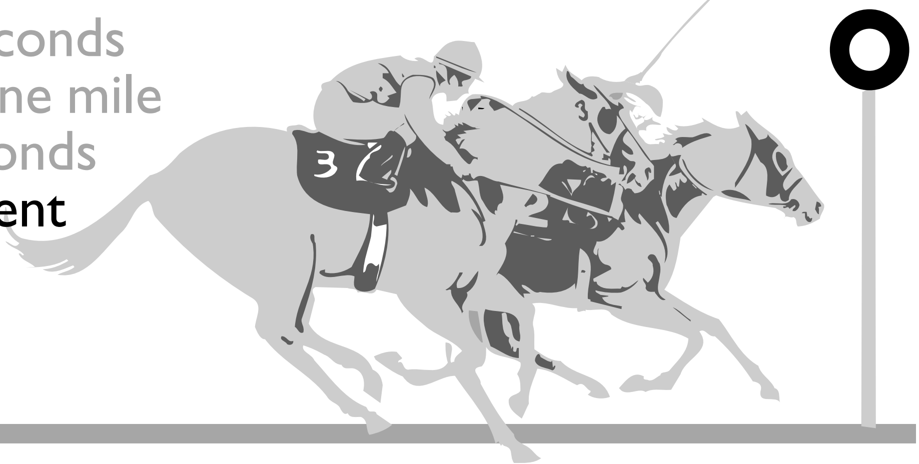
Horses for Courses

20

ALL thoroughbred horses
from **3 Arab stallions**
and **assorted mares**.

Horses for courses, **breed best to best** and **hope for the best**, ‘thoroughbred’?

Derby 1.5 Miles,
1900 - 2 minutes 42 seconds
1995 - 2 minutes 32 seconds
Improved 10 seconds
Converted to one mile
Improved 7 seconds
6.6% improvement



Humans
Mile, IAAF era
1913 - 4 minutes 14.4 seconds
1993 - 3 minutes 44.4 seconds
Improved 30 seconds
11.8% improvement

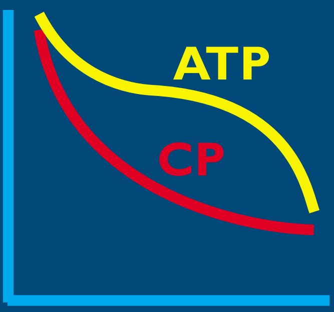
APPENDIX 1

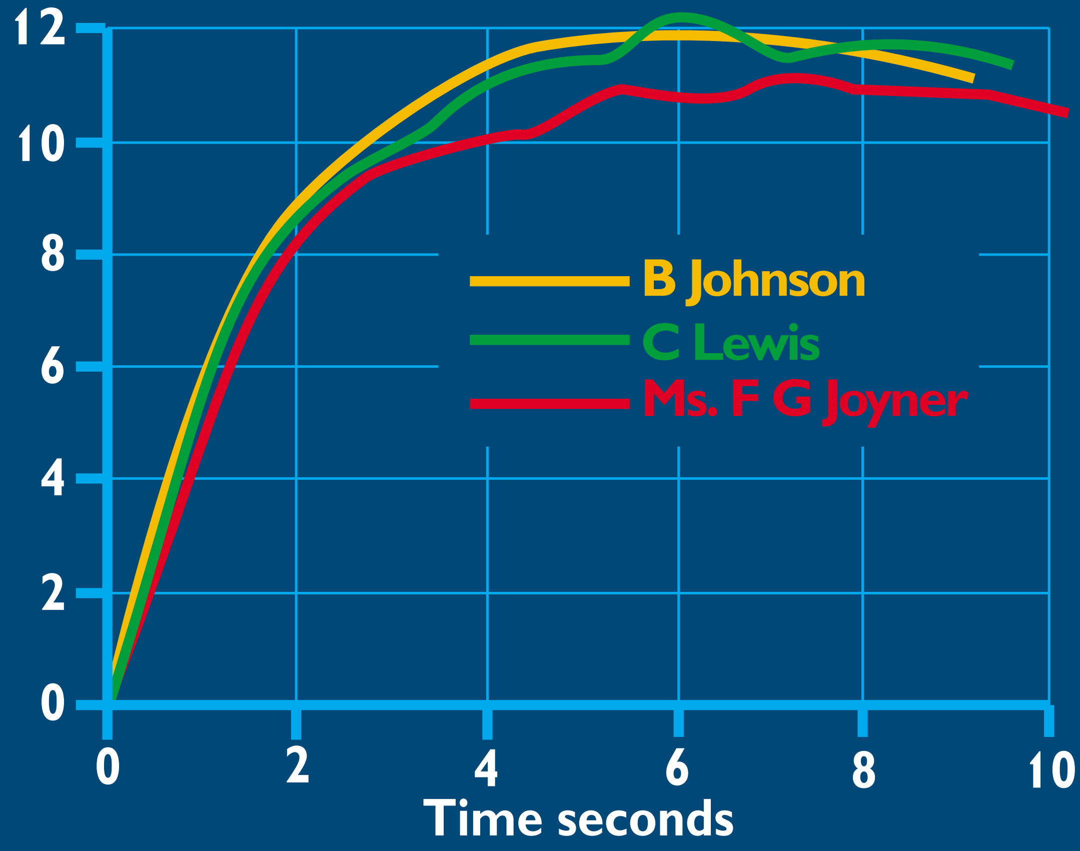
100 metres Olympic Finals

1

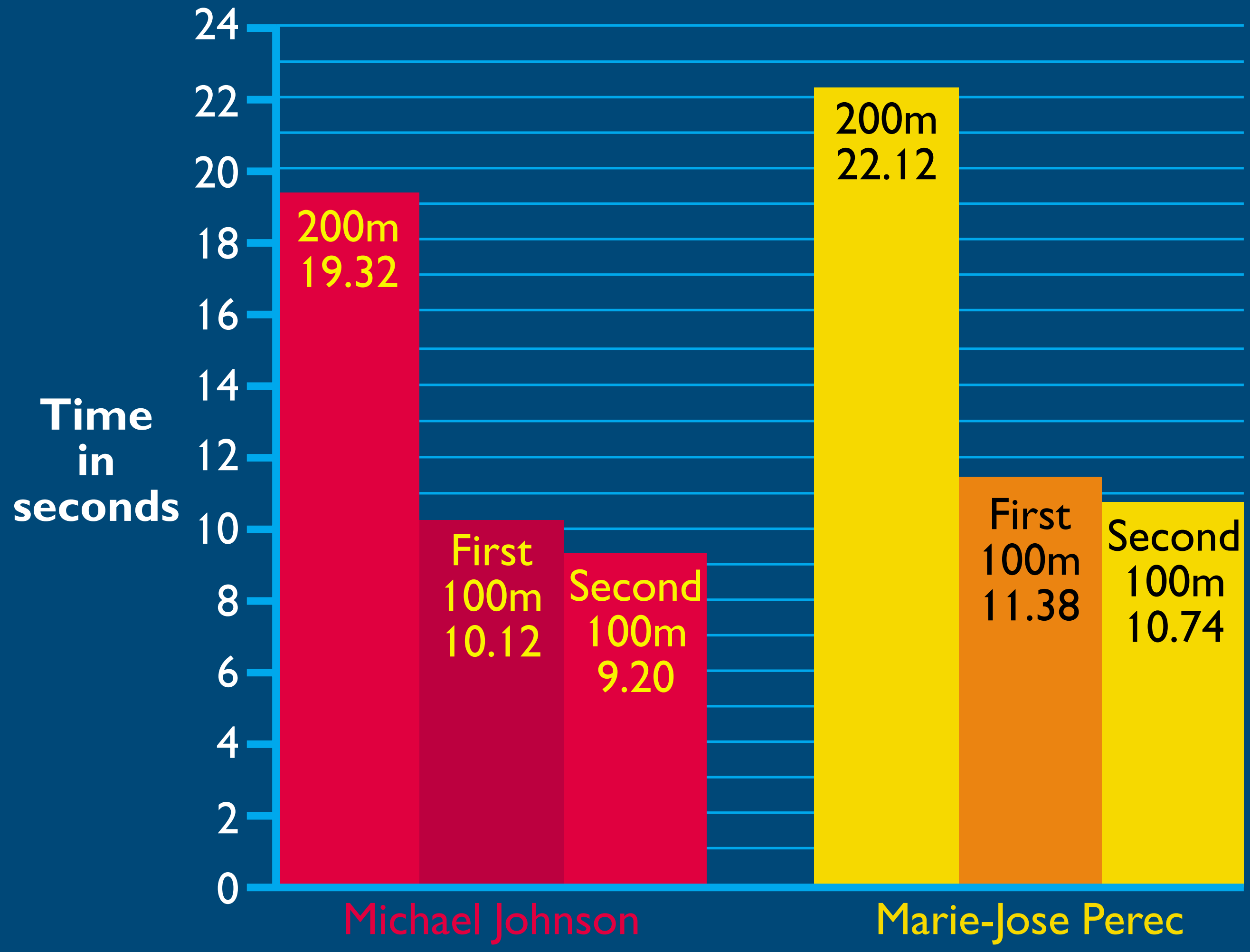
Data from IAAF Biomechanical Analysis Programme 1988 Seoul Olympics 100m Finals

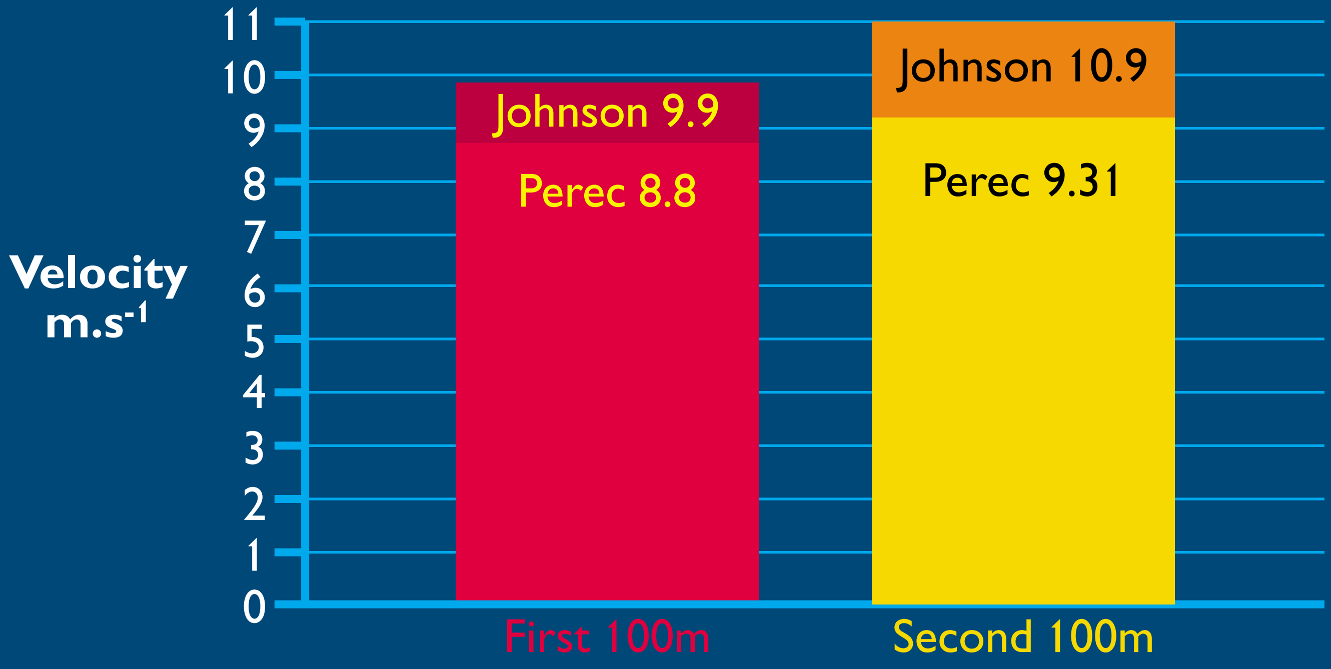
Distance metres	B Johnson seconds	C Lewis seconds	Ms. F G Joyner seconds
0	0.00	0.00	0.00
10	1.83	1.89	2.00
20	2.87	2.96	3.09
30	3.80	3.90	4.09
40	4.66	4.79	5.06
50	5.50	5.65	5.97
60	6.33	6.48	6.89
70	7.17	7.33	7.80
80	8.02	8.18	8.71
90	8.89	9.04	9.62
100	9.79	9.92	10.54





200 metres


Time in seconds
24
22
20
18
16
14
12
10
8
6
4
2
0
200m 19.32
First 100m 10.12
Second 100m 9.20
200m 22.12
First 100m 11.38
Second 100m 10.74
Michael Johnson
Marie-Jose Perec


Velocity m.s-1
11
10
9
8
7
6
5
4
3
2
1
0
Johnson 9.9
Perec 8.8
Johnson 10.9
Perec 9.31
First 100m
Second 100m

400 metres World Records *(Men)* 3

Accumulative 100m splits

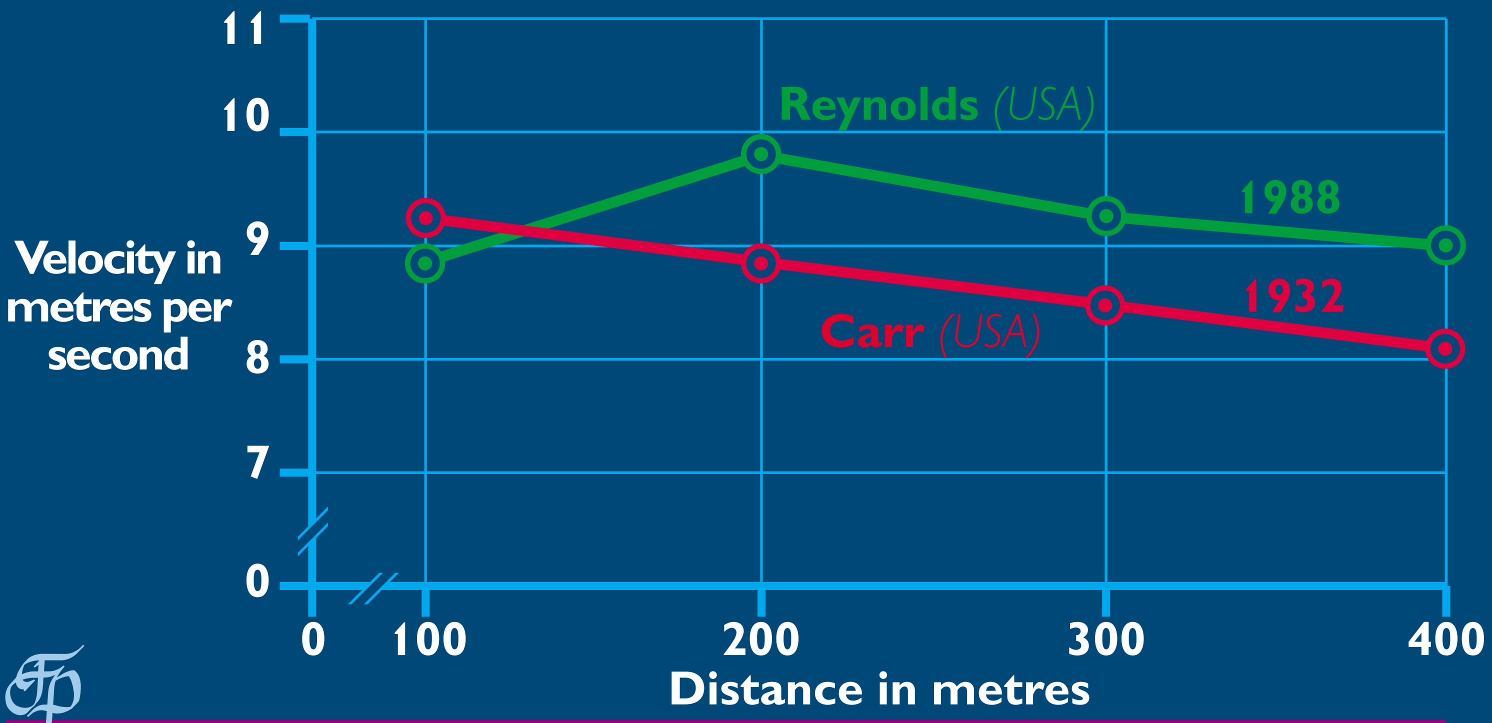
Name	Date	100 m	200 m	300 m	400 m
Carr *(USA)*	1932	10.90	22.10	33.80	46.20
Harbig *(Germany)*	1939	11.30	22.00	33.60	46.00
Klemmer *(USA)*	1941	10.80	21.80	33.40	46.00
Smith *(USA)*	1967	11.00	21.70	33.50	44.50
Evans *(USA)*	1968	10.40	21.10	32.20	43.86
Reynolds *(USA)*	1988	11.20	21.40	32.20	43.29

Times for each 100m

						Even Pace
Carr *(USA)*	1932	10.90	11.20	11.70	12.40	11.55
Harbig *(Germany)*	1939	11.30	10.70	11.60	12.40	11.50
Klemmer *(USA)*	1941	10.80	11.00	11.60	12.60	11.50
Smith *(USA)*	1967	11.00	10.70	11.80	11.00	11.13
Evans *(USA)*	1968	10.40	10.70	11.10	11.66	10.97
Reynolds *(USA)*	1988	11.20	10.20	10.80	11.09	10.82

Velocity in metres per second *(rounded up)* for each 100m

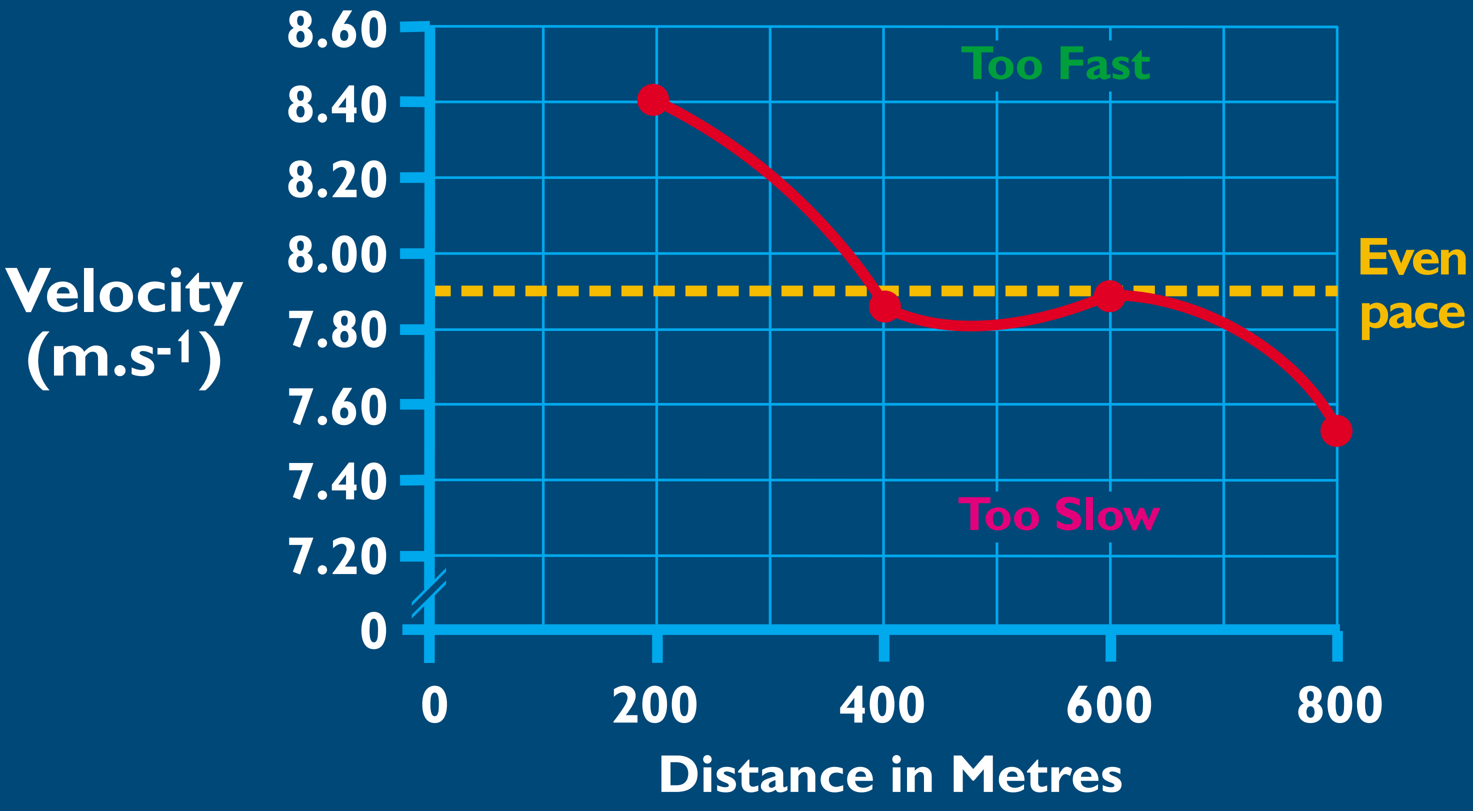
						Even Pace
Carr *(USA)*	1932	9.2	8.9	8.5	8.1	8.7
Harbig *(Germany)*	1939	8.9	9.3	8.6	8.1	8.7
Klemmer *(USA)*	1941	9.3	9.1	8.6	7.9	8.7
Smith *(USA)*	1967	9.1	9.3	8.5	9.1	9.0
Evans *(USA)*	1968	9.6	9.3	9.0	8.6	9.1
Reynolds *(USA)*	1988	8.9	9.8	9.3	9.0	9.2



800 metres World Record (Men) 4

Men's 800m World Record Kipketer 1.41.11

Distance	200m Splits Seconds	Velocity $(m.s^{-1})$
Even pace	25.28	7.91
200m	23.8	8.40
400m	25.5	7.84
600m	25.3	7.90
800m	26.5	7.55



Seb Coe's 800m World Record of 1m 41.73 in 1981 lasted until....

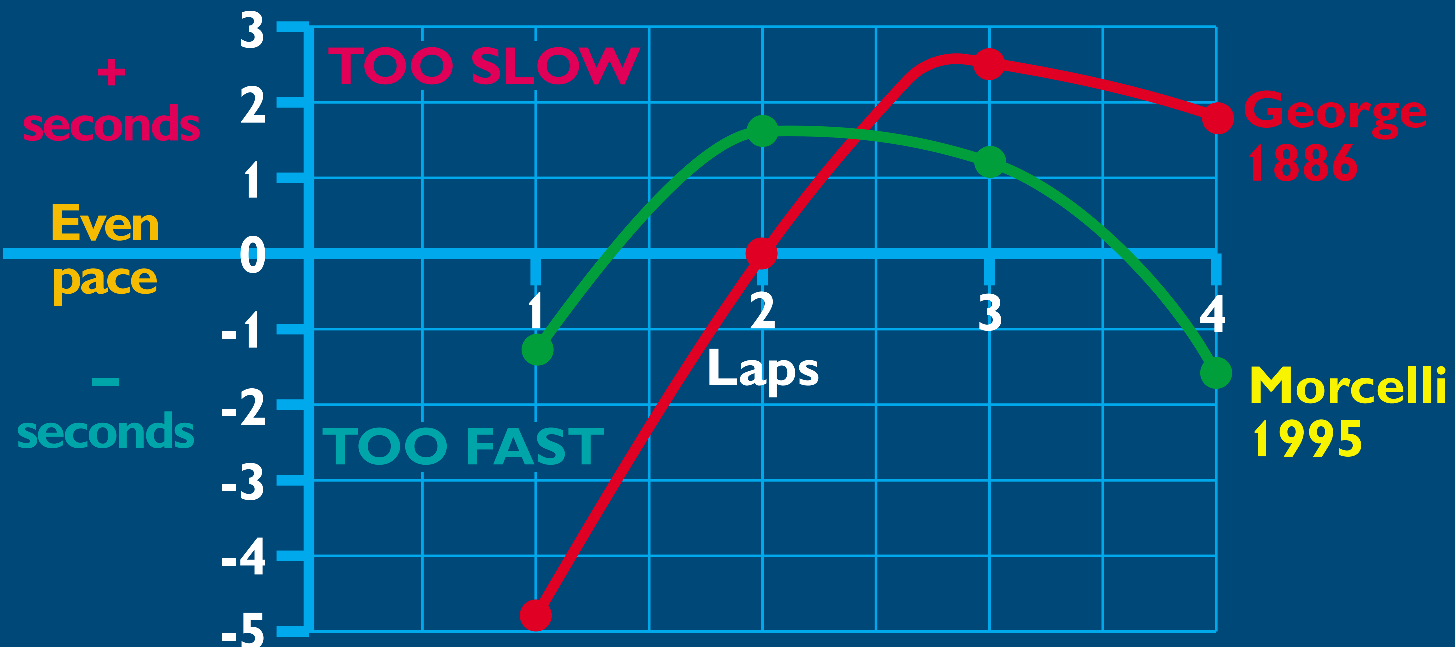
1997 when broken by Kipketer *(Kenyan)* **1m 41.11**

16 years to improve by 0.62s.

Mile World Records

Men's Mile Record & Lap *(440 yards)* Times at approximately10 year intervals

	Time	Lap 1	Lap 2	Lap 3	Lap 4	Even pace
1886 George	4.12.75	58.50	63.20	65.75	65.00	63.18
1915 Taber	4.12.60	58.00	67.00	68.00	59.60	63.15
1923 Nurmi	4.10.40	60.30	62.90	63.50	63.70	62.60
1933 Lovelock	4.07.60	61.40	62.20	65.10	58.90	61.90
1943 Anderson	4.02.60	58.50	61.50	63.50	60.60	60.65
1954 Bannister	3.59.40	57.50	60.70	62.30	58.90	59.85
1964 Snell	3.54.10	56.40	57.70	60.20	59.80	58.53
1975 Bayi	3.51.00	56.90	59.70	58.70	55.70	57.75
1985 Cram	3.46.32	57.50	57.40	58.40	53.00	56.58
1995 Morcelli	3.44.39	54.90	57.70	57.30	54.50	56.09



1953 Bannister ran first sub 4 minute mile.

1997 Kohmen *(Kenyan)* ran two sub 4 minute miles one after the other in a two mile race.
Sustained the output for twice the distance.

1886 George ran mile in 4m 12.8s
Current men's world record is now 3m 44.39s.

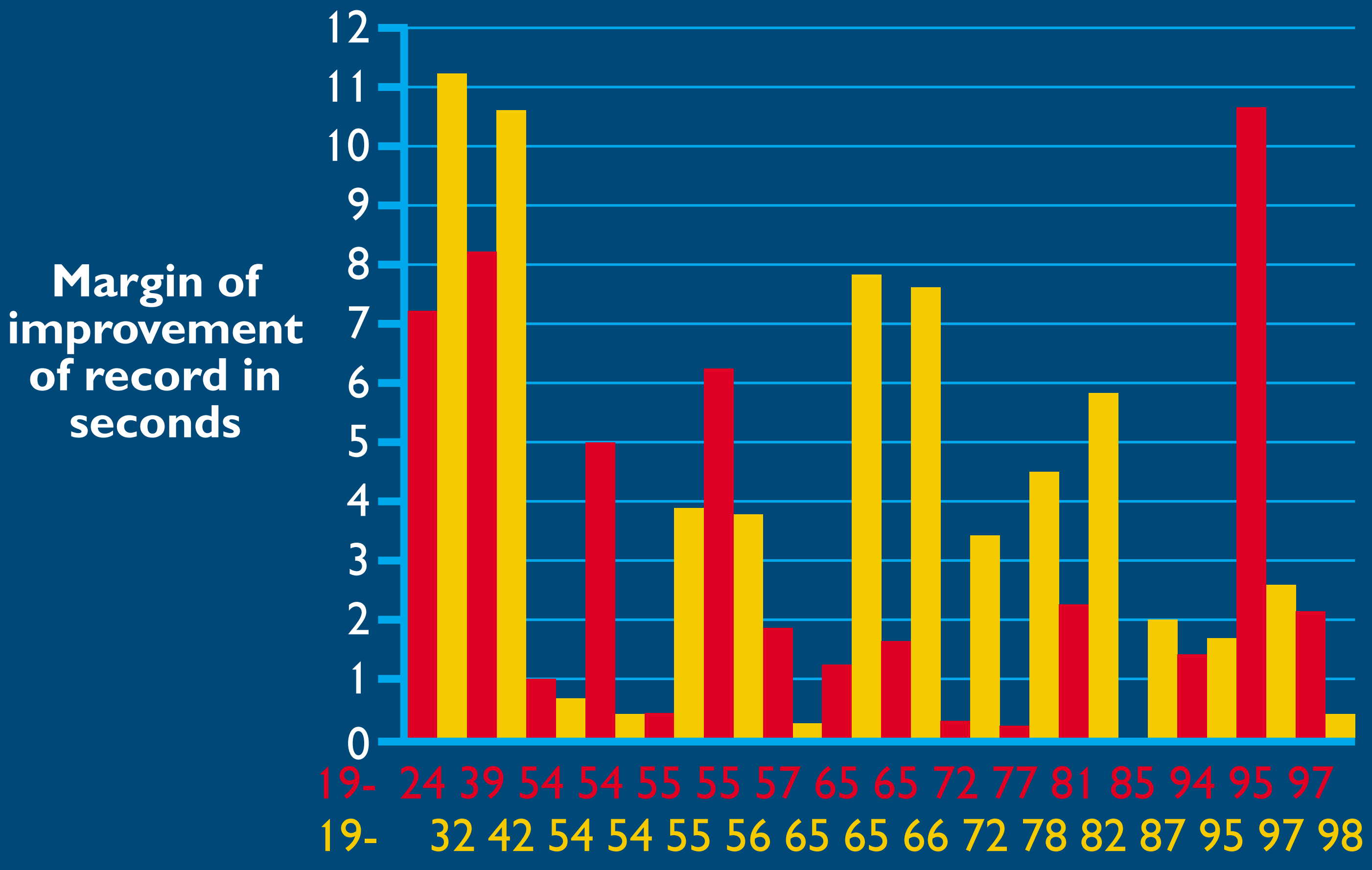
George's time of 1886 not beaten by a woman until:

Masterkova in 1996 4m 12.56s

5000 metres World Records (Men)

1 kilometre splits in selected World Records

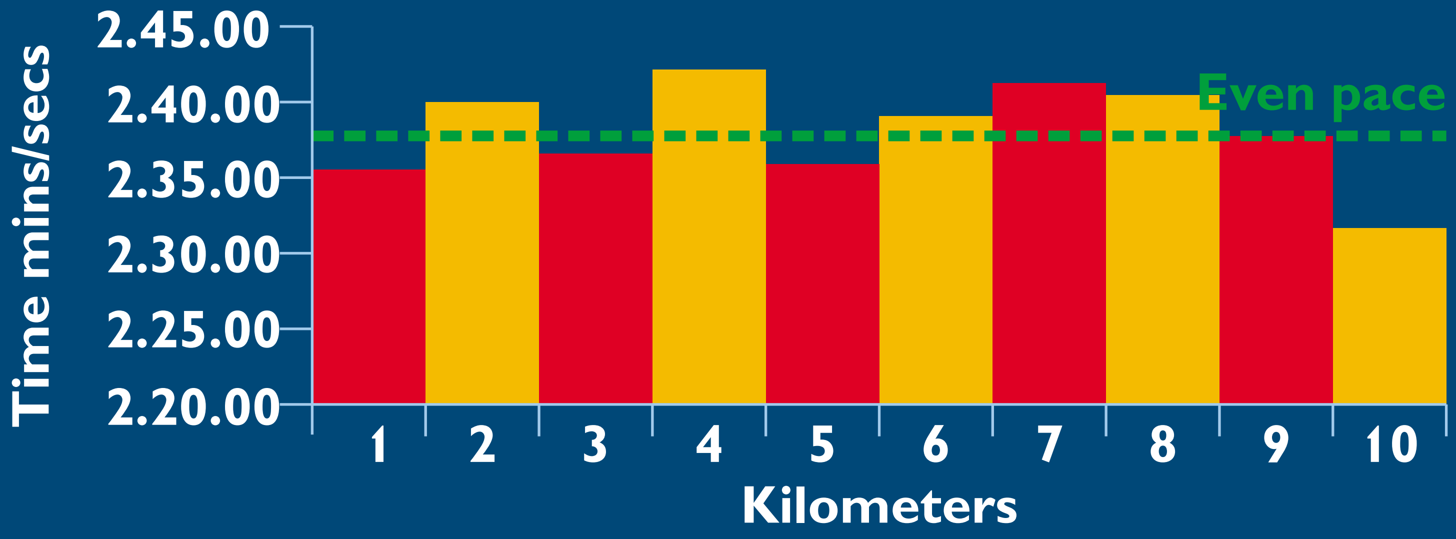
Time	Name	Year	1k	2k	3k	4k
14:36.60	**Kolehmained** *(Finland)*	1912	-	5:47.00	8:46.00	-
14:35.40	**Nurmi** *(Fin)*	1922	2:43.00	5:36.10	8:35.80	11:37.70
14:17.00	**Lehtinen** *(Fin)*	1932	2:46.50	5:40.50	8:36.00	11:33.50
14:08.80	**Maki** *(Fin)*	1939	2:46.00	5:39.00	8:32.50	11:24.50
13:58.20	**Hagg** *(Sweden)*	1942	2:40.00	5:27.00	8:18.50	11:09.00
13:57.20	**Zatopek** *(Czech)*	1954	2:47.10	5:34.10	8:23.50	11:13.40
13:46.80	**Kuts** *(USSR)*	1955	2:42.00	5:30.00	8:16.00	10:06.00
13:36.80	**Pirie** *(GB)*	1956	2:36.00	5:22.00	8:09.00	10:57.00
13:25.80	**Clarke** *(Australia)*	1965	2:39.10	5:20.40	8:04.10	10:48.70
13:16.60	**Clarke** *(Australia)*	1966	2:40.20	5:16.40	7:57.40	10:39.00
13:00.41	**Moorcroft** *(GB)*	1982	2:38.00	5:12.60	7:50.20	10:28.70
13:00.40	**Aouita** *(Morocco)*	1985	2:35.14	5:13.82	7:51.00	10:32.16
12:58.39	**Aouita** *(Morocco)*	1987	2:35.35	5:13.03	7:46.37	10:26.05
12:44.39	**Gebreselassie** *(Ethiopia)*	1995	2:34.04	5:00.70	7:42.92	10:14.15
12:39.74	**Gebrselassie** *(Ethiopia)*	1998	2:43.80	5:06.40	7:43.80	10:12.10



10 000 metres (Men)

Gebreselassie 10 000m - time 26.22.75

	'Splits'	Km times	Km pace	Even pace
First kilometer		2.35.36		Km 2.38.28
		5.15.53	2.40.17	Laps 63.31s
		7.53.03	2.37.50	
		10.35.03	2.42.00	Equivalent mile pace 4.15.28
First 5 000m	13.11.53	13.11.53	2.36.50	
		15.50.97	2.39.44	
		18.32.45	2.41.48	
		21.12.92	2.40.47	Velocity of 6.32 m·s^{-1}
		23.51.53	2.38.61	
Second 5 000m	13.11.22	26.22.75	2.31.22	



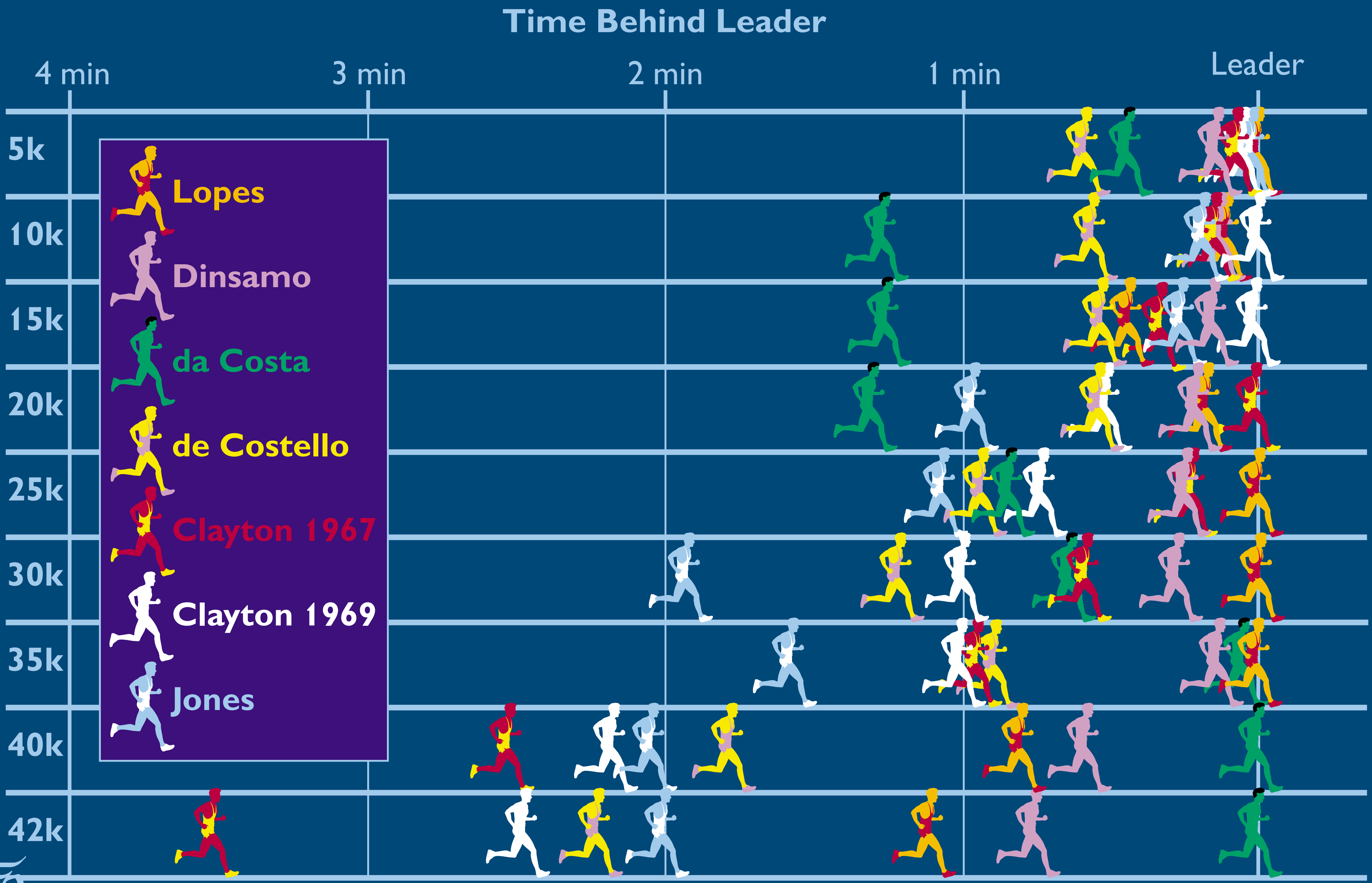
5 000m splits 13.11.53 & 13.11.22 both better than Olympic 5 000m record up until 1978.

Both 5 000m splits of Gebreselassie's 10 000m world record would have been a 5 000m world record up until 1978 when Rono ran 13.08.04

Both 5 000m splits would have also won all Olympic 5 000m finals except for:
1984 Aouita 13.05.59 O.R., & 1996 Niyongabo 13.07.96.

Although of course Olympic finals are tactical.

Marathon (42k or 26.2 miles) Men



Parity of women in ultra distance events.

Cycling

In 1967 Beryl Burton riding in the same event as the men overtook and beat Mick McNamara who was setting a new national record in the men's 12 hour time trial championship.

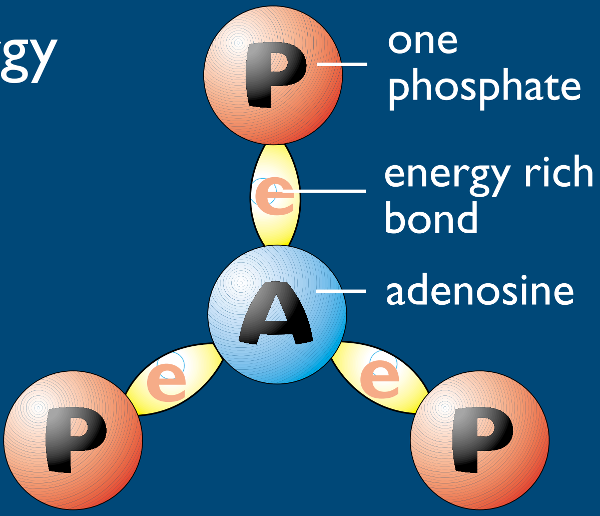
1967 Best All Rounder (BAR) Competition

	50 Miles	100 Miles	12 Hours
Men's **Mick McNamara**	1.52.25 26.66 mph	3.54.45 25.56 mph	276.52 Miles 23.04 mph
Women's **Beryl Burton**	1.56.00 25.86 mph	4.04.50 24.49 mph	277.25 Miles 23.10 mph

In the same year the fastest men's 25 mile time trial was 52 mins 55 secs *(not part of men's BAR)* = 28.35 mph while Beryl Burton's was 56 mins 7 secs = 26.73 mph

Energy from ATP

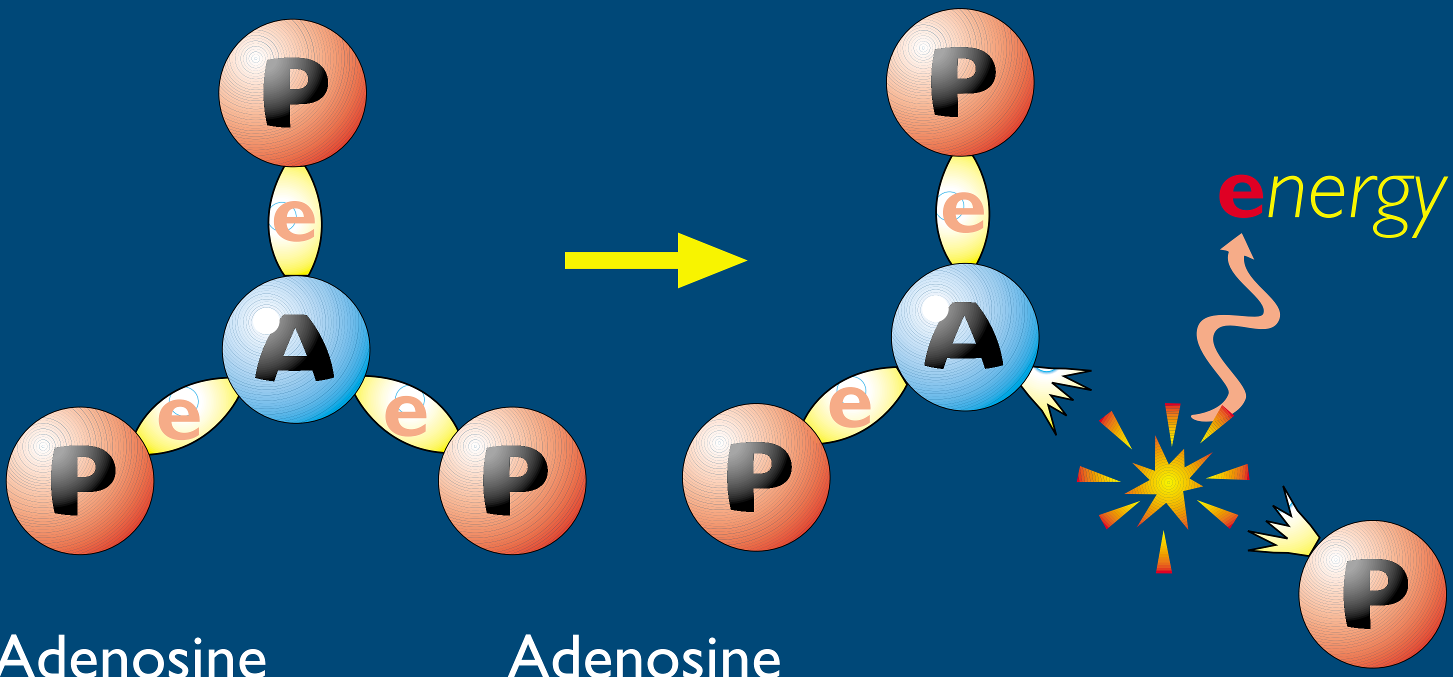
The body can only use energy in the form of **ATP**.


P
one phosphate
e
energy rich bond
A
adenosine
e
e
P
P

Adenosine **T**ri **P**hosphate has 3 phosphate groups.

If a phosphate group is removed from **ATP, energy** is released and **ADP** is formed.

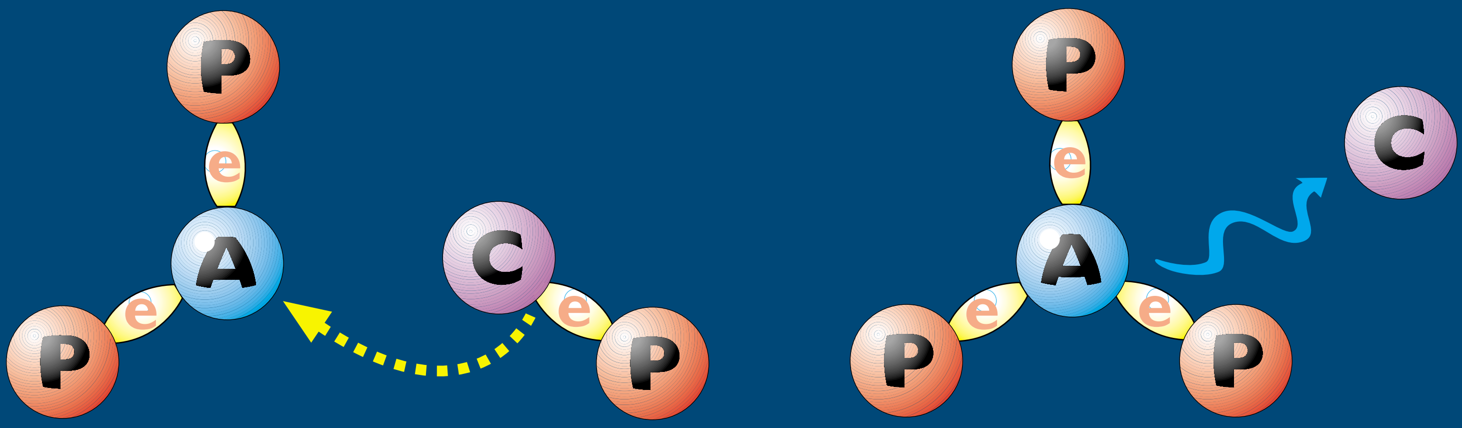
ADP has **2 phosphate** groups.


P
e
A
e
e
P
P
P
e
A
e
P
energy
P

Adenosine **Tri** Phosphate *(ATP)* → Adenosine **Di** Phosphate *(ADP)* + Phosphate *(P)* + Energy

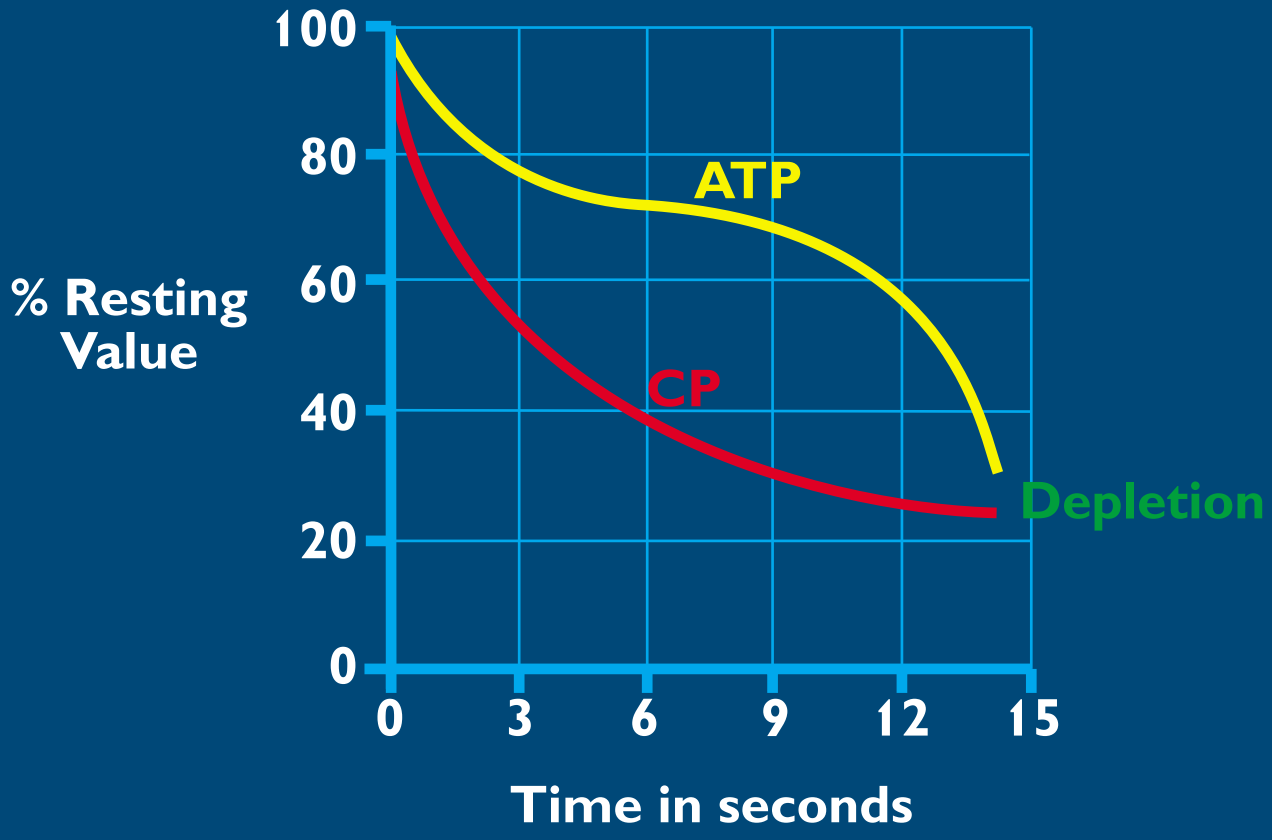
ATP Regeneration from Creatine Phosphate (CP)

The body only contains enough ATP for a few seconds of flat out activity
Then the ATP must be regenerated from the ADP using an energy reserve substance Creatine Phosphate (CP)



Adenosine **Di** Phosphate *(ADP)* + **Creatine Phosphate *(CP)*** → Adenosine **Tri** Phosphate *(ATP)* + **Creatine**

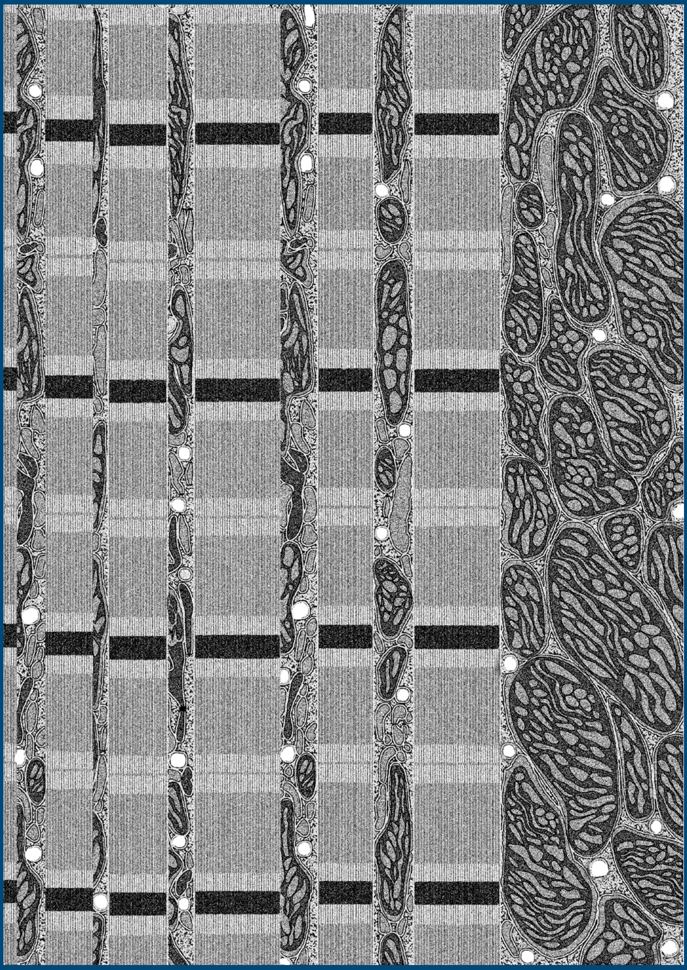
Levels of ATP and CP during a flat out sprint



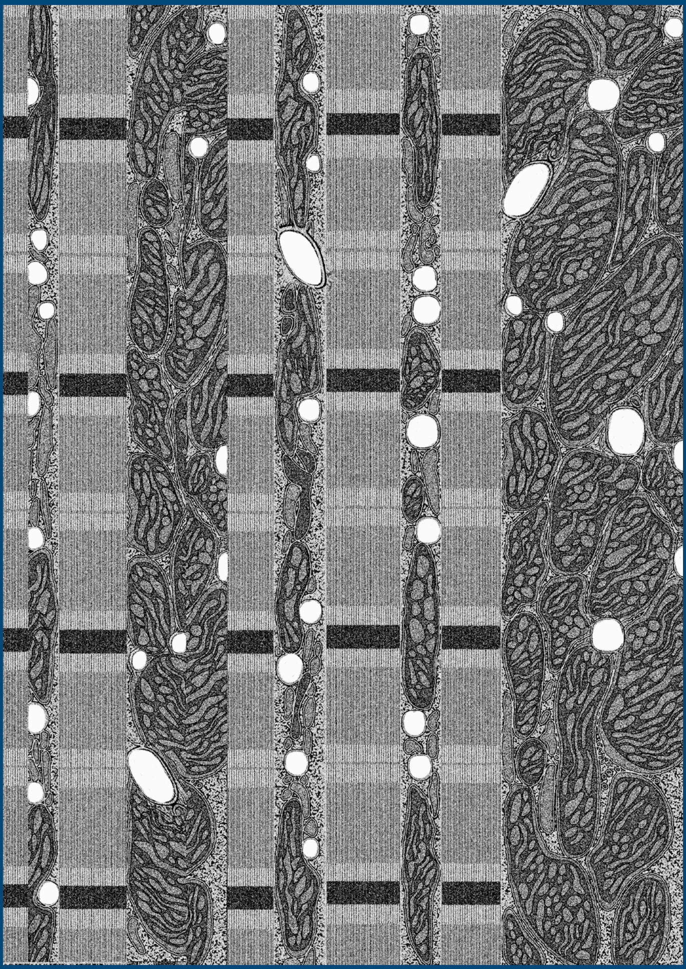
Skeletal Muscle Myofibrils as Seen Under the Electron Microscope

12

Untrained

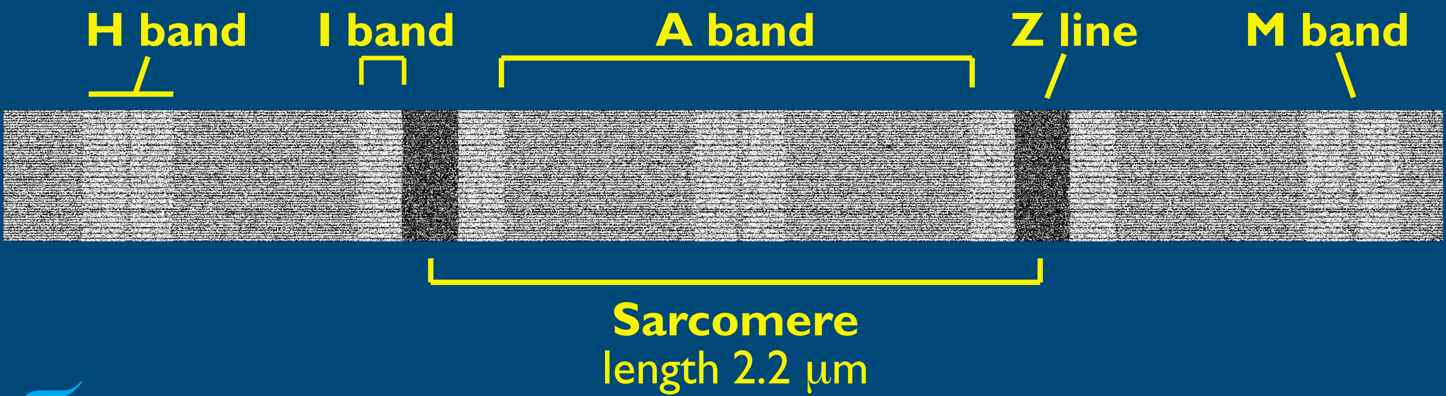


Trained

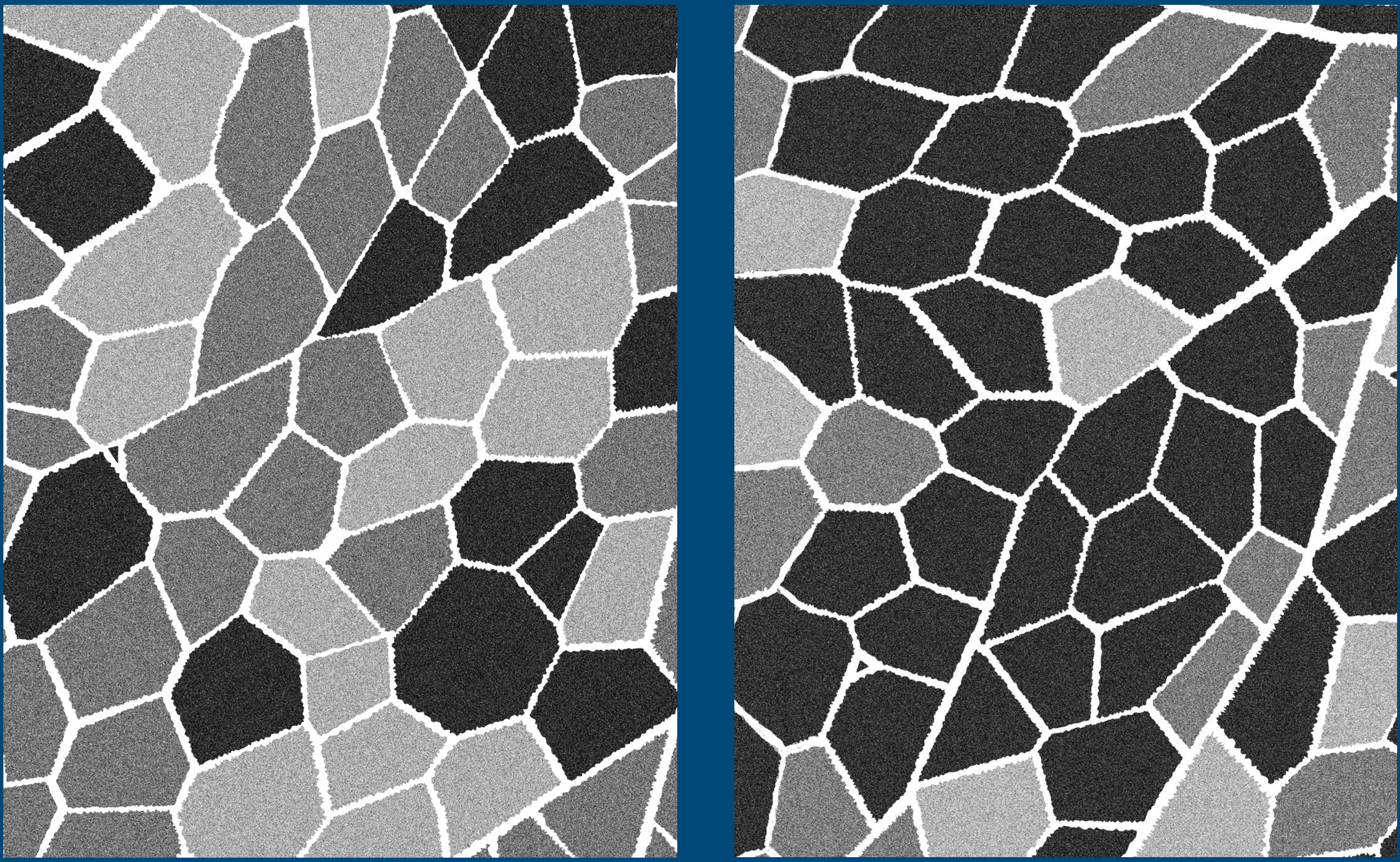


Note the increased size and number of mitochondria and fat vacuoles in the endurance trained muscle.

Magnification x5000


H band
I band
A band
Z line
M band
Sarcomere
length 2.2 µm

Slow and Fast Twitch Muscle Fibres in Humans



Sprint Runner
70% Fast-twitch fibres *(light)*

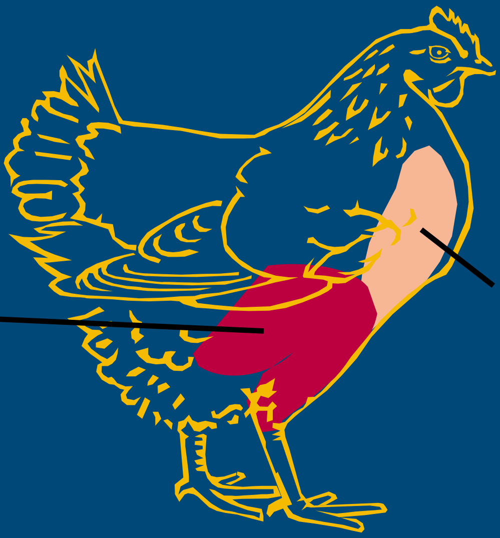
Marathon Runner
90% Slow twitch fibres *(dark)*

Skeletal Muscle Fibre Types and their Properties

Type	Colour	Contraction	Metabolism	Properties
I	Red	Slow-twitch	Oxidative (aerobic)	Fatigue resistant Low force/power
IIA	White	Fast-twitch	Oxidative/glycolytic (aerobic/anaerobic)	Intermediate
IIB	White	Fast-twitch	Glycolytic (anaerobic)	Fatigue susceptible High force/power

Slow & Fast Twitch Muscle Fibres in Other Animals

Chicken

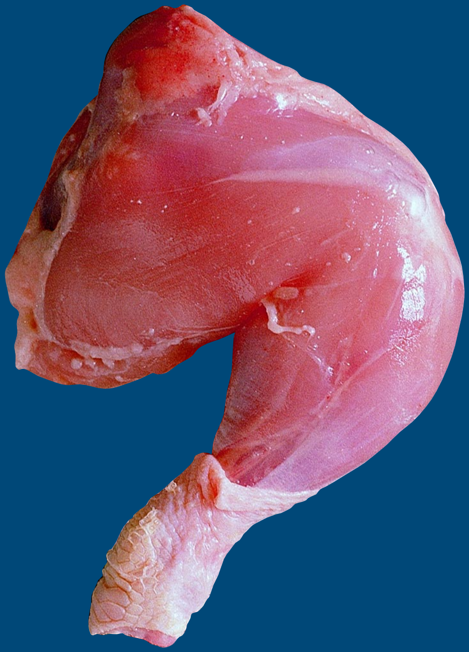


Thigh muscles of leg

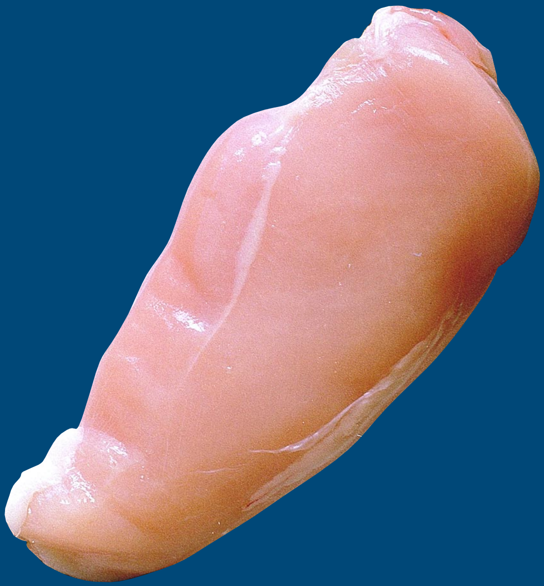
All red slow twitch muscle fibres

Pectoral flight muscles

All white fast twitch muscle fibres



Thigh muscles of leg



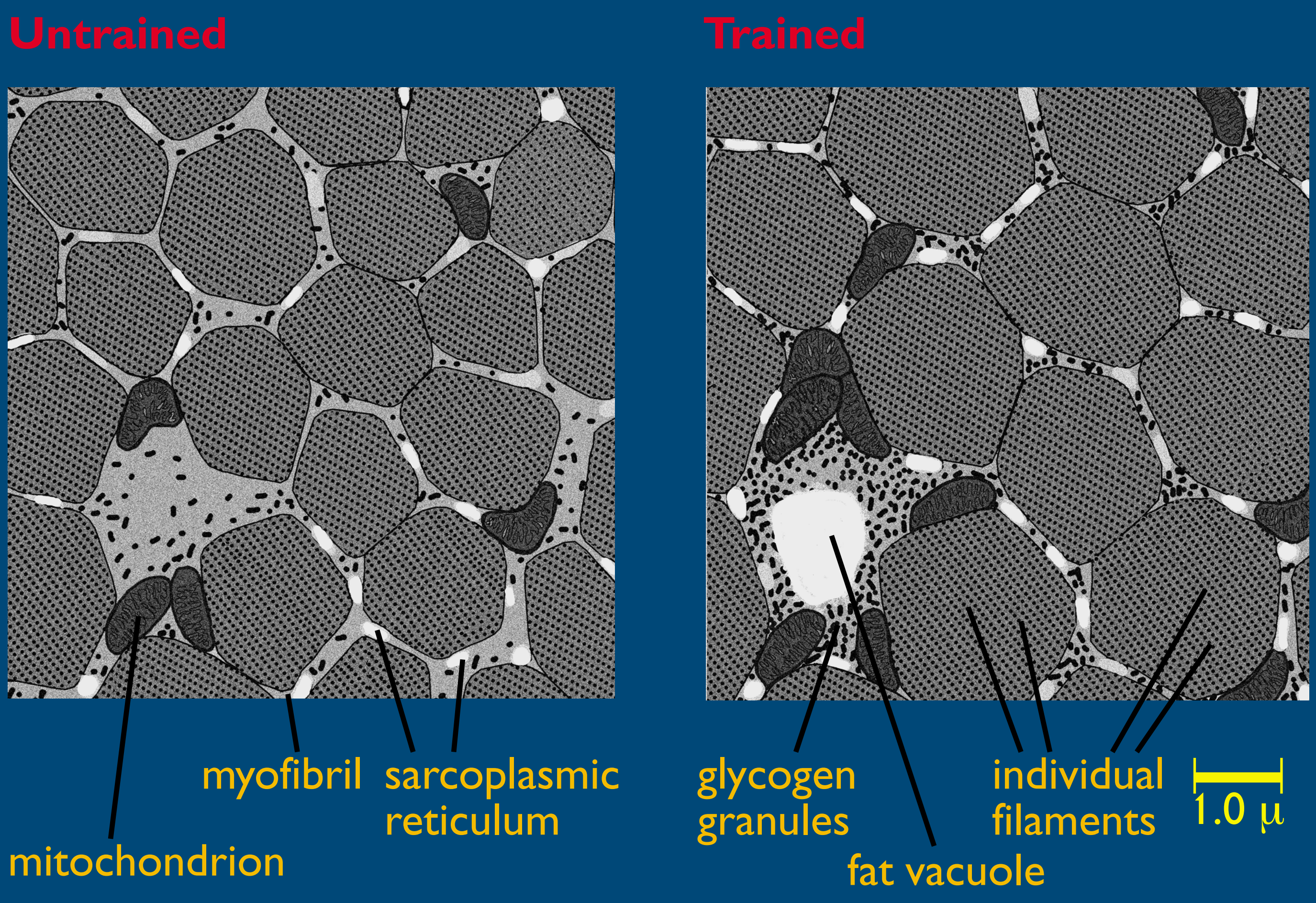
Pectoral flight muscles

Blood supply in fresh meat adds a reddish tinge to both.
The bright red colour of meat in the window of the butcher's shop
is due to an artificial additive to make it more 'attractive'.

Effect of Endurance Training on Slow Twitch Muscle Fibres

15

Effect of aerobic training on type I slow twitch muscle fibres. Mitochondria, myoglobin, glycogen stores, fat vacuoles, all increase. The figure shows these changes amongst the myofibrils within a single muscle fibre.


Untrained

Trained

mitochondrion
myofibril
sarcoplasmic reticulum
glycogen granules
fat vacuole
individual filaments
1.0 µ

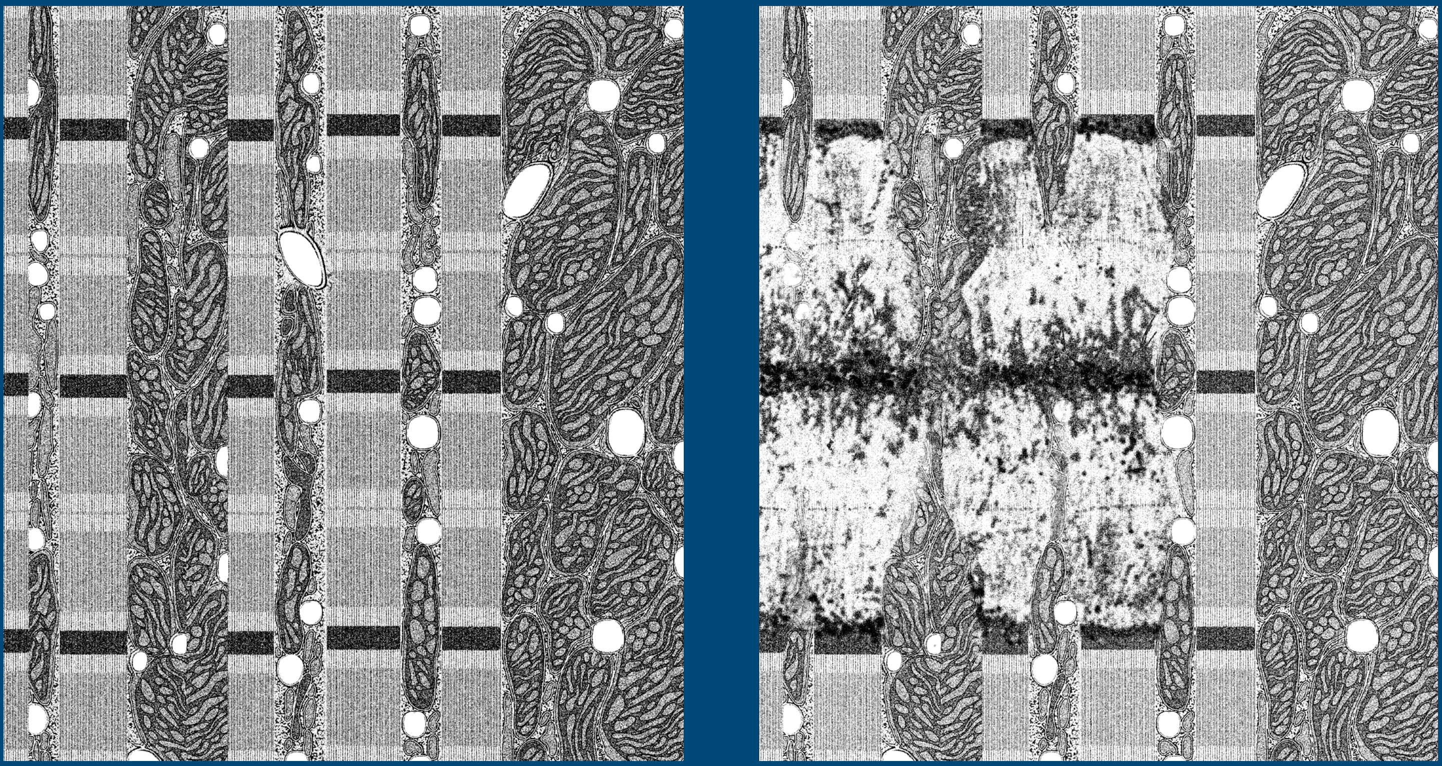
Myoglobin - Average untrained 2g/100g muscle.

Myoglobin - Endurance trained 7g/100g muscle.

= 250% increase

Muscle Damage and Delayed Onset Muscle Soreness

Muscle damage after strenuous endurance work



Rested muscle

Muscle damaged by hours of running

Magnification x 5000

Note breakdown of integrity of fibres as a result of membrane damage.

Much of the damage caused by running occursduring the eccentric phases eg. qudriceps whilst running downhill.

Such damage results in an inflamatory reaction by the immune system.

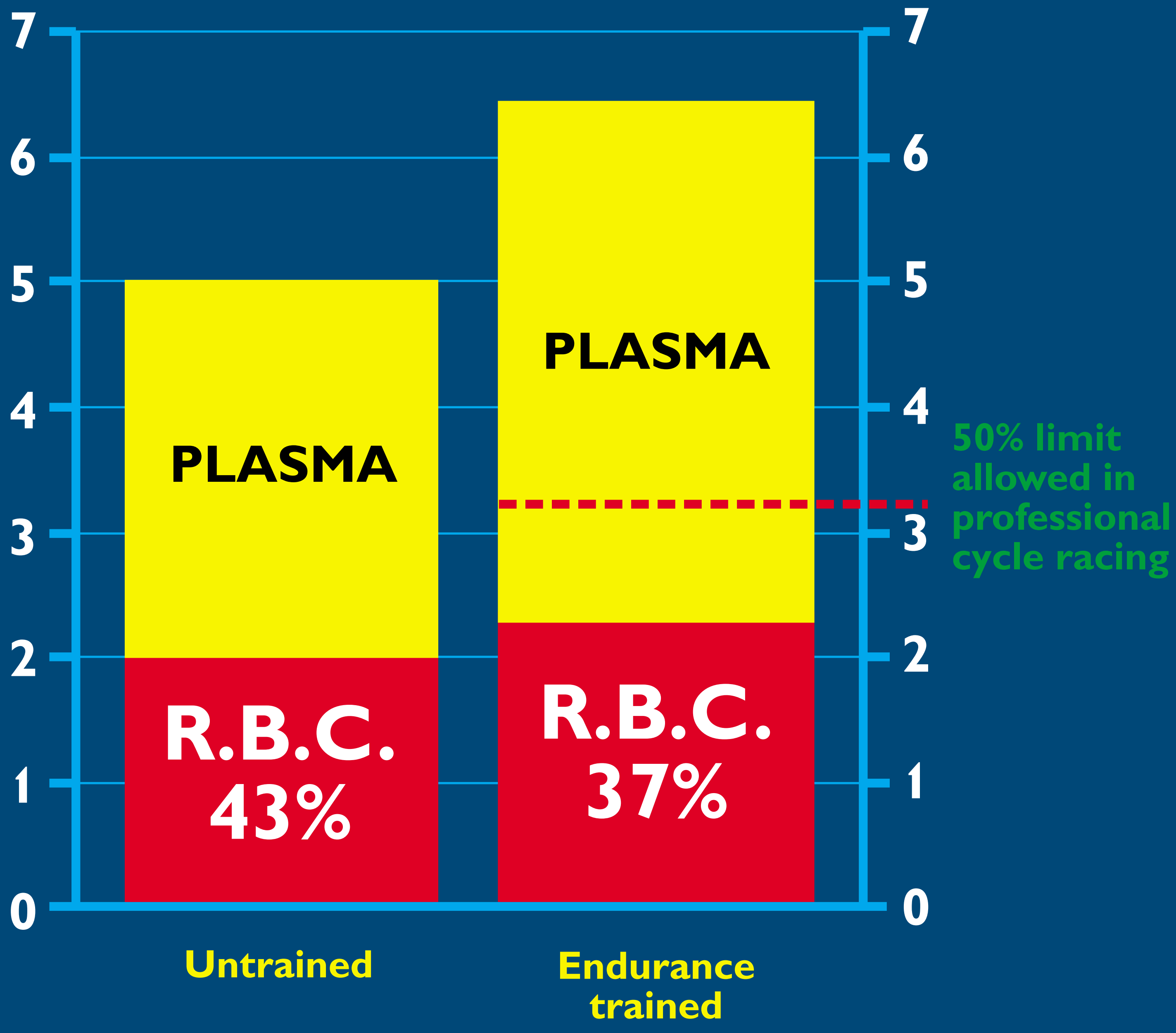
Effects of Endurance Training & Doping on Blood Composition

17

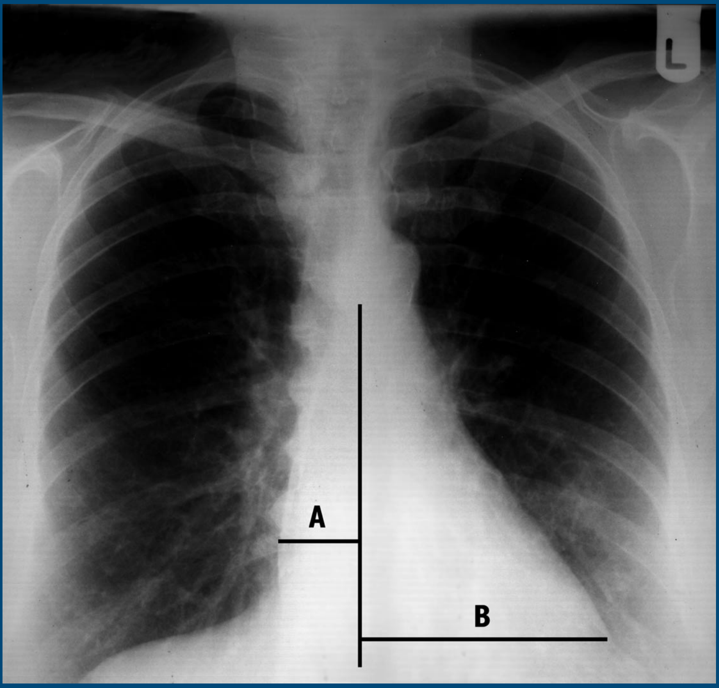
Endurance Athletes can experience Pseudo-anaemia

Due to increased plasma volume

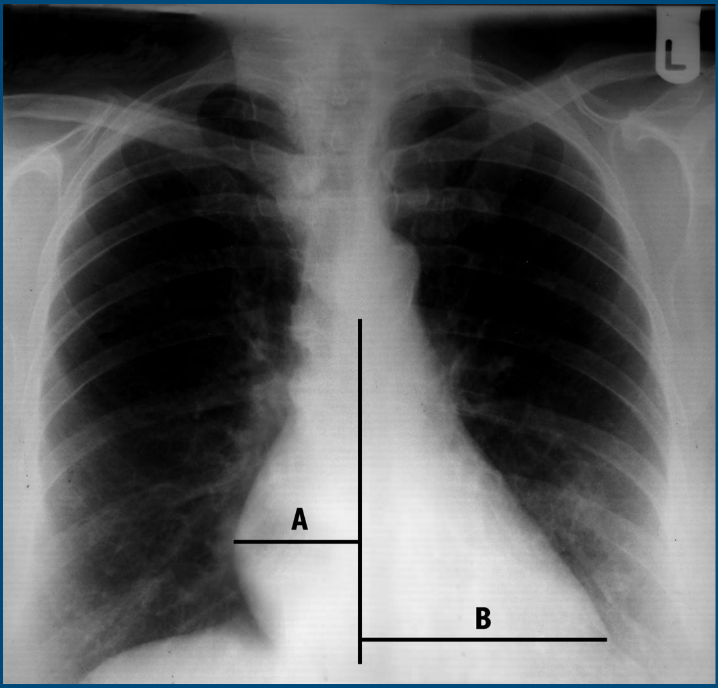
Conversely high red blood cell counts also occur



Heart Untrained and Trained



Average Male Heart Size



Endurance Athlete Heart Size

The cardiothoracic ratio is the sum of lines A and B divided by the largest transverse diameter of the chest (normally 0.50)

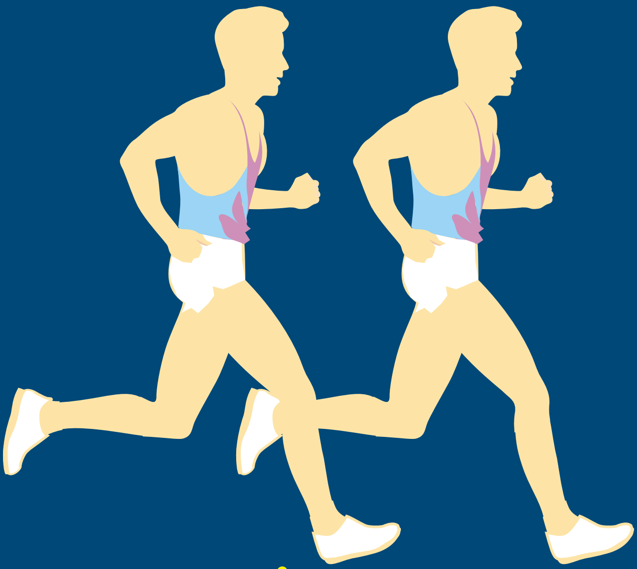
$$\text{Cardiac Output } (\text{cm}^3.\text{min}^{-1}) = \text{Heart Rate (bpm)} \times \text{Stroke Volume } (\text{cm}^3)$$

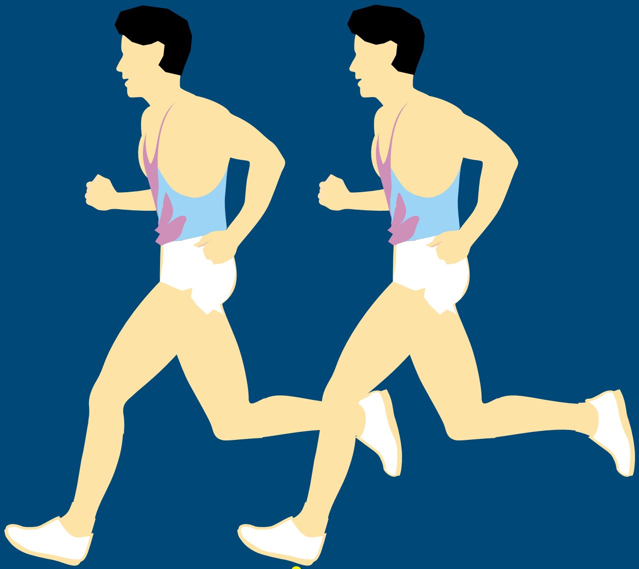
	Normally Active Male	Elite Male Marathon Runner
Resting Heart Rate (bpm)	78	32
Maximum Heart Rate (bpm)	188	180
Heart Rate Range (bpm)	110	148
Reasting Stroke Volume of left ventricle (cm^3)	75	183
Maximum Stroke Volume of left ventricle (cm^3)	80	166
Resting Cardiac Output (cm^3)	5850	5850
Maximum Cardiac Output (cm^3)	15040	29880
Heart Weight (g)	300	350
Total Heart Volume	800	1600

Nature or Nurture- Twin Studies

10 pairs of Monozygote Twins

Fully standardised and laboratory controlled training programme for 20 weeks





Relative $\dot{V}O_2$ max gain

+1 +3

Average

+2

Relative $\dot{V}O_2$ max gain

+15 +17

Average

+16

Members of the same pair make similar improvements but there are large differences between the different pairs

Horses for Courses

ALL thoroughbred horses
from **3 Arab stallions**
and **assorted mares.**

Horses for courses, **breed best to best** and **hope for the best**, ‘thoroughbred’?

Derby 1.5 Miles,
1900 - 2 minutes 42 seconds
1995 - 2 minutes 32 seconds
Improved 10 seconds
Converted to one mile
Improved 7 seconds
6.2% improvement

Humans
Mile, IAAF era
1913 - 4 minutes 14.4 seconds
1993 - 3 minutes 44.4 seconds
Improved 30 seconds
11.8% improvement